UNIV OF
TORONTO
LIBRARY

University of Pennsylvania

CONTRIBUTIONS

FROM THE

Department of Neurology

AND THE

Laboratory of Neuropathology

FOR THE YEARS 1913 AND 1914

(REPRINTS)

VOLUME VII

UNIVERSITY OF TORONTO LIBRARY

146813
30/8/18

PHILADELPHIA

CONTENTS.

The Cerebral Mechanism of Emotional Expression.....Charles K. Mills, M.D.
Discussion of Dr. Mills' Paper on the Cerebral Mechanism of Emotional Expression.
George McClellan, M.D., James Hendrie Lloyd, M.D., Smith Ely Jelliffe, M.D., William G. Spiller, M.D., Augustus A. Eshner, M.D., and J. Cardeen Cooper, M.D.
The Relation of the Myopathies.....................William G. Spiller, M.D.
Segmental Distribution of Spinal Root Nucleus of the Trigeminal Nerve.
Andrew H. Wood, M.D.
Punctiform Hemorrhagic Pigmentation of the Cerebral Pia Mater.
Edward Mercur Williams, M.D.
The Effect of the Removal of the Hypophysis in the Dog.
Joshua Edwin Sweet, M.D., and Alfred Reginald Allen, M.D.
Paraplegia in Hodgkin's Disease—Treatment by Laminectomy and the Roentgen Rays..............Charles K. Mills, M.D., and Edward Martin, M.D.
The Direct Ventro-lateral Pyramidal Tract............William G. Spiller, M.D.
Histological Changes of the Central Nervous System Produced by Heloderman Venom.......................................Samuel Leopold, M.D.
Remarks on the Histopathological Changes in the Spinal Cord due to Impact. An Experimental Study.......................Alfred Reginald Allen, M.D.
The Mental Manifestations of Epilepsy...................N. S. Yawger, M.D.
Syphilitic Tests in Latent and Treated Syphilis.
E. P. Corson-White, M.D., and S. D. Ludlum, M.D.
Complete Bilateral Isolated Paralysis of the Seventh Nerve Developing Four Months After the Primary Infection of Syphilis...........Harvey M. Ewing, M.D.
Some Causes of Disappointment in Operations on Brain Tumor.
William G. Spiller, M.D
A Consideration of Some of the Phases of the Insanity Problem.
John H. W. Rhein, M.D.
The Treatment of the Insane in Psychopathic Wards...John H. W. Rhein, M.D.
The Present Status of the Insanity Problem..........John H. W. Rhein, M.D.
Neurology at the Recent (Seventeenth) International Congress of Medicine. Impressions with Comments on Some of the More Important Subjects Presented.
Charles K. Mills, M.D.
Apraxia in Relation to Aphasia......................John H. W. Rhein, M.D.
Presidential Address before the American Psychopathological Association.
Alfred Reginald Allen, M.D.
Paramyoclonus Multiplex, Including a Case with Necropsy Showing Lymphocytic Infiltration of the Pia..................Edward Mercur Williams, M.D.
Experience with Crotalin at the Oakbourne Epileptic Colony...N. S. Yawger, M.D.
Alcoholism and Epilepsy, Also So-called Acute Alcoholic Epilepsy.
N. S. Yawger, M.D.
A Comparison of the Onset and Character of the Apoplexy Caused by Cerebral Hemorrhage and by Vascular Occlusion....Williams B. Cadwalader, M.D.
Hallucinations of Hearing and Diseases of the Ear ....John H. W. Rhein, M.D.
Extensive Bilateral Subdural Hemorrhage over Both Cerebral Hemispheres without Motor Paralysis or Irritation..................William G. Spiller, M.D.

CONTENTS

Silas Weir Mitchell, M.D., LL.D. His Place in Neurology. Charles K. Mills, M.D.
Tuberous Sclerosis and Other Unusual Features of Epilepsy. . . N. S. Yawger, M.D.
Acute Myelitis, Secondary to Perirectal Abscess, Developing a Few Hours after Severe Fright. . . . . William G. Spiller, M.D.
Symmetrical Pain in the Heel from Exostosis of the Os Calcis. William G. Spiller, M.D.
Cerebral Palsies Without Demonstrable Anatomical Findings. John H. W. Rhein, M.D.
The Different Theories of Aphasia. . . . . Charles K. Mills, M.D.
Contusion of the Brain. . . . . John H. W. Rhein, M.D.
Tumor of the Crus Cerebri . . . . John H. W. Rhein, M.D.
Aneurisms of the Vessels of the Brain. . . . . John H. W. Rhein, M.D.
Cerebellar Symptoms and Cerebellar Localization, Including Kinematographic Observations on Cerebellar Phenomena. Charles K. Mills, M.D., and T. H. Weisenburg, M.D.
Bilateral Oculomotorius Palsy from Softening in Each Oculomotorius Nucleus. William G. Spiller, M.D.
The Polyneuritic Form of Acute Poliomyelitis: A Clinical and Pathologic Study. S. Leopold, M.D.
Muscle Tonicity, Emotional Expression, and the Cerebral Tonectic Apparatus. Considered Especially with Reference to a Case of Bilateral Caudato-lenticular Degeneration. . . . . Charles K. Mills, M.D.
Spontaneous Subarachnoid Hemorrhage. A Contribution to the Subject of Meningeal Hemorrhage. . . . . Samuel Leopold, M.D.
Progressive Lenticular Degeneration. . . . . Williams B. Cadwalader, M.D.
Psychoses Following Apoplexies. . . . . John H. W. Rhein, M.D.

Reprinted from the Transactions of the College of Physicians of Philadelphia, 1912

THE CEREBRAL MECHANISM OF EMOTIONAL EXPRESSION[1]

By CHARLES K. MILLS, M.D.

PROFESSOR OF NEUROLOGY IN THE UNIVERSITY OF PENNSYLVANIA

LITERATURE

EMOTION and emotional expression can be approached from several standpoints, as from biology, anatomy, physiology, psychology, pathology, and physiognomy. The organic basis of emotional expression in man, which will be in considerable part the subject of the present contribution, includes the neural and muscular periphery, the structural correlatives, spinal and bulbar, of this periphery, and the controlling and regulating cerebral and cerebellar, cortical and subcortical centres and tracts concerned. While central mechanisms will in the main be discussed attention will be directed to other elementary features of the subject in order to take a comprehensive glance at the whole.

No attempt will be made to present a complete study of the literature of emotion and emotional expression, for which the reader is referred for enlightenment on different phases of the subject to such well-known authors as Sir Charles Bell, Charles Darwin, Hughlings Jackson,[2] and the school of localizationists,

---

[1] This paper as presented at the College of Physicians was illustrated by numerous lantern slides and moving pictures, a list of which is appended to the article. The moving pictures which were exhibited under the direction of Dr. T. H. Weisenburg include cases considered in the paper and others chosen for the purpose of general demonstration.

[2] Besides Hughlings Jackson, the school of localizationists embraces many names like those of Hitzig, Ferrier, Charcot, Horsley, Dana, and so many others whose work is well known that no specific reference need be made to any. In addition to the literature referred to in the article a partial bibliography is appended.

James, Irons, Wundt, Lavater, Mantegazza, von Bechterew, Nothnagel, von Monakow, Brissaud, Lewandowsky, Obersteiner, Mingazzini, and Bianchi. In the elucidation of the phenomena of emotional expression the original work in artistic anatomy of Dr. George McClellan, of Philadelphia, holds a place of first importance.

Among my immediate colleagues excellent work has been done by Spiller, Lloyd, and Weisenburg, on the subject of the central disorders of emotional expression, especially in articles on pseudobulbar paralysis and diseases of the great basal ganglia.

In 1907 Dr. Spiller collaborated with me in the production of an article on the symptomatology of lesions of the lenticular zone, in which the pathology of involuntary laughing and weeping was incidentally considered.

PARALYSIS OF EMOTIONAL EXPRESSION IN MAN

The clinical history of a case in which emotional paralysis of one side of the face was the striking feature was recorded by me in 1908. This patient died in October, 1911, and in February of the present year I published a preliminary note on a new symptom-complex which included emotional paralysis of one side of the face, this contribution containing an abstract of the microscopic findings and a *résumé* of the clinical history of this case. It is only necessary to repeat here that the features of the syndrome were marked ataxia of the upper and lower extremities on the left side, with paralysis of emotional expression in the face, nerve deafness, and loss of the sense of pain, heat, and cold on the right. The pathological examination showed destruction of the ventral portion of the dentate nucleus and adjacent superior cerebellar peduncle. The upper two-thirds of this peduncle were degenerated, and as a consequence the right nucleus ruber was at least one-third smaller than the left. The left mesencephalic root of the fifth nerve and left lateral lemniscus were also much degenerated.

The cause of the loss of emotional expression in the face, while as yet not thoroughly determined, would appear to be related to the destruction of afferent tracts in the lemniscus, or of this and some part of an important cerebellar arc.

Recorded examples of emotional paralysis like the above are rare, the reason for this being twofold. In the first place, such loss of expression either in the face or limbs or in both is in fact infrequent as the result of disease, and especially disease of the central nervous system; and secondly, this form of impairment or loss of control is doubtless at times overlooked, the investigator's attention being usually confined to a study of voluntary movement.

Spiller has published with photographs a case in which partial emotional paralysis on the same side was present, the case being one of tumor of the cerebellopontile angle. He inclines to the view that the partial emotional palsy was dependent upon interference with the cerebellar arc by way of the posterior cerebellar peduncle and dentate nucleus. Two cases reported by Nothnagel are well known, although owing to the size of the lesions which involved various parts besides the thalamus and adjoining regions, the inference as to a thalamic centre of mimetic expression, according to the views of von Bechterew is not as sure as Nothnagel and others who have made use of these cases believe. While the thalamus doubtless plays a part on the afferent side of the bulbocerebral mechanism of expression we are not yet in position to decide as to the exact expressional role of this ganglion, but this subject will be considered somewhat more fully later in the paper.

EMOTIONAL EXPRESSION IN LOWER ANIMALS AND IN MAN

The muscles of expression in the face of the lower animals differ considerably from those of man, both in accordance with the general form of the animal's head and face and with its power of emotional expression. Carnivorous and graminivorous animals, as Bell has shown, differ considerably in their facial musculature. In the former these differences are such as to make special variations in expression, particularly as indicated in showing the teeth and in the use of the eyes.

New muscles in the face are added as animals rise higher and higher in the scale. This is illustrated by a comparison of the anthropoids and man with the commoner carnivorous and graminivorous animals. Among the added muscles are those particularly concerned in expression, as the corrugator supercilii and the triangularis oris. In the anthropoids these muscles while present are not as finely developed for the expression of emotion as in man.

For the purpose of the present discussion it must be constantly borne in mind that the nervous mechanism of emotional expression in the lower animals is less independent of the will and shows marked points of difference from the nervous mechanism of emotion in the human being. Emotion as apparently expressed by these animals is usually more immediately related with what they are doing or are about to do. While it may be instinctive or reflex it is not conditioned as much as in man by the presence of an independent emotional system.

EMOTIVE CORTICAL ZONE (MIDFRONTAL OR PREFRONTAL)

In the early years of the modern era of cerebral localization it was held that the motor zone of the cortex extended for an equal or almost equal distance, both cephalad and caudad of the central fissure. As time elapsed, however, experimentation upon the lower animals, more especially upon the anthropoids, and the more careful observation and interpretation of clinicopathological phenomena, demonstrated that this area was largely, if not altogether, in front of the Rolandic fissure. As shown in the scheme by the writer, first published in 1905, in a paper written in conjunction with Dr. Frazier, it will be seen that the motor zone thus represented is largely confined to the precentral convolution, having, however, a projection or extension forward into the second, and superior portion of the third frontal convolution. In the anthropoids it is even more confined to the precentral convolution than in man.

As animals have ascended in the scale, new centres of representation have developed as evolutional outgrowths of the original motor zone. In the case of the human being such development has always taken place as man has added to the movements necessary for existence, those especially related to acts of skill and emotional expression. Such development has been toward the anterior pole of the brain. Centres for writing and for speech are in part the result of this forward expansion, but with these I am not particularly concerned in the present communication.

In lower animals, as has been so beautifully demonstrated by Darwin, emotion is expressed by movements of various parts of the body, although the face is not entirely excluded from such manifestations. In man facial movements are preëminently the means of expression, and cortical centres concerned with the representation of movements of expression must exist, as such expression is not always involuntary, and even when known to be non-volitional, has had recently or remotely a voluntary origin.

Experimental faradization of the human midfrontal cortex during the performance of craniectomies of considerable size has shown that the movements evoked are such as are commonly used in facial emotional expression, although except in a few instances not confined to the purposes of expression.

Some of the movements obtained by such electrical excitation were those of turning the head to one side, of forward bending of the head, of closure of both eyes (other observers having noted opening of both eyes), and of opening and shutting movements of the jaws and mouth. The results thus obtained were from the faradization of cortical regions in advance, and some of them much in advance, of the precentral convolution, in which most of the unilaterally differentiated movements are represented as has been indicated by various methods of investigation. Movements of the jaw and of opening the mouth seem to have a large area of representation well forward in the motor region.

Recently, during the search for a tumor, the frontal lobe from the central fissure to within about one to two inches of the anterior pole of the brain was exposed by Dr. Frazier. Unipolar faradization at a point in the second frontal convolution, near the junction of its middle with its anterior third, caused elevation slightly toward the median line of the inner angle of the eyebrow on the side of stimulation and elevation of the eyelid. During the same operation stimulation farther back caused depression of the angle of the mouth and twitching of the platysma on the opposite side. Movements of the eyebrows and eyelids are a part of those concerned with the expression of grief.

It may be interesting to observe here in connection with the effect obtained of elevating one eyebrow by stimulating the prefrontal cortex of the same side that an individual occasionally has an unusual facility in the volitional performance of this movement. A well-known Russian actress sometimes makes use of this movement in changing her facial expression. The movement in the human being seems to be ipsilateral as demonstrated by our experiment, but it may also be a contralateral movement or a part of one that is bilateral. Many of the movements of expression are of course as is well known bilateral, the centre of one side governing the movements of both.

The centre for the corrugator supercilii is doubtless situated in the immediate vicinity of the point, faradization of which resulted in elevation of the inner angle of the eyebrow. The movements of the face which express distress, pain, or anguish are of late development in the process of evolution and probably have their centres of representation situated in advance of all other motor centres.

In these experiments I was searching for the centres for the movements of what Darwin has aptly called the grief muscle, believing these on theoretical grounds to be in the most anterior portion of the motor zone, or more probably cephalad of this zone as usually described. Darwin explains ~~describes the muscles which give~~ the peculiar appearance of the central portion of the face between and above the eyes as due to the contraction of the orbiculars of the eyelids, the pyramidals of the nose and the corrugator. Darwin holds that the movements of these muscles are apparently checked by the more powerful action of the central fasciæ of the frontal muscle. The movement due to this fascia on the side of application was the one obtained in our experiment.

Owing to the exigencies of the operation it was deemed best not to increase or continue the stimulation, but I have no doubt if this had been done in the same vicinity the movements of the corrugator supercilii, orbicularis palpebrarum, pyramidalis nasi, and the frontalis would have been obtained on both sides. In other words, in the region where fragments of this general movement were determined the whole could have been obtained by sufficient stimulation.

Duchenne, who is cited by Darwin, differs somewhat from the latter, believing that it is the corrugator which raises the inner corners of the eyebrows and that this is antagonistic to the upper and inner part of the orbicular muscle as well as to the pyramidalis nasi. It is likely that it may eventually be found that centres for all the central and bilateral movements of the face concerned with expression are located in what we have chosen to term the emotive zone or area.

The part played by the posterior extremity of the third frontal convolution in speech has interest in this connection, as the first attempts at speech by primitive man were probably in response to emotions called out by pain, hunger, fear, or other causes.

HISTRIONIC AND SPONTANEOUS LAUGHING AND WEEPING AND OTHER ALLIED FORMS OF EMOTIONAL EXPRESSION

The spontaneous movements of laughing or weeping are regarded as among the most automatic, instinctive, or reflex of the movements of expression. The mechanism for voluntary movements seems to have but little share in the phenomena. The ability voluntarily to imitate laughing, weeping, frowning, and other more or less marked expressions of emotion, without or with the presence of such emotion, must be brought about through some association between the voluntary and involuntary mechanisms. Borée, an Alsatian actor, was photographed in many poses intended to indicate the different emotions. These were collected in a volume of 119 autotypes, representing crying, care or anxiety, ~~and~~ laughing, etc. One thing which seems evident in making a comparison of histrionic and spontaneous emotional expression is that the former has a forced element not present in the latter, in which an appearance of relaxation predominates.

With Dr. Weisenburg, with the aid of moving pictures, I studied the simulation of various emotions by well-trained actors, comparing the expressions produced with those exhibited in spontaneous laughing and weeping as illustrated by actual cases and by photographic reproductions of Darwin and others. However, skilful the reproduction a difference between the two manifestations is more or less apparent, a difference which is probably determined in large degree by the manner in which the voluntary motor mechanism is associated or dissociated from that for so-called involuntary movements. Whether emotional expression is ever absolutely divorced from voluntary movement may be doubted, its discussion involving perhaps some consideration of the theories which hold that movement precedes emotion, or the reverse.

EFFECTS OF MUTILATION OR DISEASE ON THE MUSCLES OF EXPRESSION

The brutal but skilful barber surgeon of the time of James II cut the muscles of the face in young children in such a way that while the entire musculature might be at rest momentarily, or even for a longer time under the strong influence of the will, the victim of this procedure was always in the imminence of grinning or wild laughter. This mutilation was in the interest of the cruel trade of supplying involuntary laughers for mountebank performances. In a case of this sort the factitious peripheral state is one in which involuntary expression could not be sufficiently controlled by the cerebral inhibitory mechanism, hence the explosions of facial movement. That the muscles under the conditions present can be controlled at all, proves the existence of an ordinarily strong and efficient inhibitory nervous apparatus.

It is an interesting point in connection with cases of irrepressible laughing and weeping of different sort that the facial innervation, and probably the innervation of other parts of the body concerned with the expression of emotion, is often weakened in the intervals between the involuntary outbreaks. I have again and again noticed this, although it is not manifest in all cases. The masked appearance of the countenance spoken of by von Monakow is sometimes in evidence as in one of the most striking instances which I have ever seen of irrepressible weeping associated with howling. This man's face when at rest reminds one of a countenance void of emotion, a dull or blank expression characterizing it. The movements in these cases are bilateral as is also the masking of the features between the outbreaks. As von Monakow well puts it the irrepressible agitation gives the impression of a ready mechanism which in normals is regulated by inhibition, finely controlled by the will, which is suddenly freed from all fetters and breaks roughly into prominence. When the musculature was ingeniously cut, in Hugo's *Man Who Laughs*, so as to put it, so to speak, as if on a knife-edge balance, the powerful internal inhibitory mechanism could not be continuously effective.

SUMMARY OF THE SITES OF ORGANIC LESIONS CAUSING DISORDERS OF EMOTIONAL EXPRESSION

Lesions very diversely situated may produce loss, exaltation, or disorder of emotional expression. Pathological evidence shows that such lesions are usually, but not always, bilateral. When unilateral, they are most frequently right-sided. These lesions may be situated (1) in the emotive cerebral cortex, or jointly in this and the motor zone as usually recognized, in which case they are bilateral or one-sided, and when unilateral, large and either very destructive or very irritative. (2) Bilateral lesions of the frontothalamic tracts (?) (3) Bilateral lesions of the frontopontile tract. (4) Bilateral lesions of the lenticula. (5) Bilateral lesions of the geniculate or corticobulbar tract of the internal capsule. Sometimes lenticular and capsular lesions are present in the same case. (6) Bilateral lesions of the thalamus (von [illegible] ~~Monakow~~ and Nothnagel) (?) (7) Bilateral or right-sided unilateral lesions of the red nucleus and cerebral peduncle. (8) Bilateral lesions or central or unilateral lesions of the pons. (9) Unilateral or bilateral lesions of the dentate nucleus and superior cerebellar peduncle and lateral lemniscus.

EMOTION AND EMOTIONAL EXPRESSION AND THEIR CORTICAL REPRESENTATION

A word or two might first be said here with regard to the mental process called emotion and some of the theories which have been advanced regarding its cerebral structural basis.

No sharp distinction can be made as some would have us believe between feeling and emotion. They are to our way of thinking but degrees of the same general process. According to the James-Lange theory of emotion the mental process so denominated succeeds rather than antedates the movements which usually express emotion, the sequence being perception or sensation, movement and emotion. If this is the case, the question of emotional expression becomes more important in the consideration of the nature and probable seat of emotion.

This question of the probable seat of the representation of emotion (and here I am not speaking of emotional expression) has not escaped the interest and attention of some neuropsychologists. According to Flechsig, for instance, the general sensorimotor cortex is the central focus or seat of the emotions and passions. He accepts von Bechterew's view of a thalamic centre for emotional expression, and believes that impulses from the sensorimotor cortex, as understood by him, are conveyed to this thalamic centre and thence to the periphery. Some have held that in the gyrus fornicatus, others that in the post-central convolution, and still others that in the thalamus itself painful sensations are represented.

An American psychologist, Mary Whiton Calkins, has attempted to formulate a theory of the localization of pleasure and pain. This will be best understood by giving it in the language of the writer in the citations which follow. The views which are apparently accepted by Calkins with regard to sensorimotor localization are not those held by me, as I hold to the separate representation of sensation and of movement in the cortex, and also that the motor area is entirely or almost entirely cephalad of the central fissure. This difference of opinion, however, does not prevent a comprehension of the hypothesis advanced as to the brain structures concerned in emotion and in its expression.

"Pleasantness and unpleasantness," says Calkins, are occasioned by the excitation of fresh or of fatigued cells in the frontal lobes of the brain, and the frontal lobe is excited by way of neurones from the Rolandic area of the brain. When the neurones (or cells) of the frontal lobes, because of their well-nourished and unfatigued condition, react more adequately to the excitation which is conveyed to them from the Rolandic area, an experience of pleasantness occurs; when, on the other hand, the cells of the frontal lobe, because they are ill nourished and exhausted, react inadequately to the excitation from the Rolandic area, then the affection is of unpleasantness; when, finally, the activity of frontal lobe cells corresponds exactly to that of the excitation, the given experience is neither pleasant nor unpleasant, but indifferent."

"But we have not completed our study of the bodily conditions of emotion," says this writer again, "until we try to discover the brain or nerve changes which condition these changes in pulse, respiration, and the rest. A probable account of these brain changes is the following. First (*a*) sensory brain centres are excited through perception or imagination of a given object; next (*b*) the excitation of these sensory neurones spreads to the brain centres of bodily sensations and movements, that is, to the region forward and back of the fissure of Rolando, and there excites motor cells. This excitation of the motor neurones of the Rolandic region is then carried (1) downward to lower brain centres in the *medulla oblongata,* which control the unstriped muscular coatings of the inner organs of the body, such as bloodvessels, heart, and intestines. In this way the internal circulatory changes are brought about: the heart-beat and pulses are checked or increased, and the arteries (not the big ones near the heart, but the smaller, thin-walled vessels in outlying parts of the body) are dilated or constricted, thus occasioning either a flush and rising temperature or pallor and chilliness. The downward excitation is carried (2) to the striped or skeletal muscles attached to the bones of the body, and thus the 'external' changes in breathing and muscular contraction are occasioned. Both sorts of bodily change, the 'internal' and the 'external,' excite end-organs of pressure, and the internal changes excite also end-organs of warmth and cold; and these excitations of the end-organs of pressure and of warmth or cold are carried upward by ingoing nerves to the sensory cells of the bodily-sensation-and-movement-centre (the Rolandic area). The excitation of these sensory cells is the immediate condition of all the organic sensations (whether due to internal changes or to external movements) which are present in emotional experience. And from the Rolandic area, excitations carried to the frontal lobe bring about that adequate (or inadequate) excitation of neurones which conditions the pleasantness (or unpleasantness) of emotions."

If either of these theories, and especially that of Calkins, be tentatively accepted, and if in addition we adopt the James-Lange theory of emotion following rather than preceding the muscular movements, in which are to be included not only the movements of skeletal muscles but also the movements of circulation, respiration, secretion, excretion, etc.—by which emotion is expressed, it would seem logical to believe that the cortical representation of emotional expression is closely related in the cerebral mantle to the cellular region of the brain in which the activities which more particularly cause emotion are themselves represented. If instead of the James-Lange theory we adopt that of Irons and many others who believe that movements follow or are closely concomitant with emotion, the same would probably be true with regard to the cortical representation of emotional expression.

I am not one of those who believe that the problem of emotion or of any other great mental process is to be explained by regarding it in some vague way as a complex expression of the action of the cerebral cortex as a whole. It may be true that the multitudinous centres, receptive, associative, ideational and emissive, take part in a general sense in all those processes which we collectively regard as mental or mind products. Nevertheless, the trend of all modern investigation as to cerebral function is in the direction of developing a belief in the centralizing or polarizing in certain more or less limited areas of the cortex the particular activities which are concerned with lower and higher mental processes—with concrete and abstract thinking.

With Bianchi and others I believe that in the frontal lobes, especially in the prefrontal region, occurs the final synthesis of those cerebral activities which result in the forms of conscious action or recognition which are included under such terms as attention, judgment, comparison, fancy, and emotion. It may be that some of these processes, as attention for instance, have also lower or special representation in centres and associative tracts more directly connected with the periphery, but a part, and a higher part, of such processes has its synthesis in this frontal or prefrontal region.

Where then is the site of the expression of emotion? A full consideration of all the phenomena associated with emotion—those of so-called spontaneous laughing and weeping in children or adults, of consciously forced emotion or emotional expression, as in acting, of such expression as the result of ideation in itself dependent upon memorial residua—cannot but lead us with our knowledge of function to the opinion that the cortex or cerebral mantle has always a place in the explanation of the phenomena. It may be urged that involuntary or spasmodic laughter or weeping such as has been so well observed and studied by numerous neurological investigators, occurring as it most frequently does because of lesions in subcortical centres and tracts, such as ~~has been~~ previously summarized, indicates that the representation of emotional expression is in these parts. But in reply to this it may be said that we have here to consider not true emotion or its representation, but the action of parts concerned subcortically, that is, unconsciously, with the expression of what appears to be emotion. It is a frequent observation by those who have studied cases of these forms of disease that the patients apparently exhibit these phenomena of involuntary laughing and weeping without any concomitant emotion, or if emotion be present it is a sequence rather than a concomitant or precedent of the involuntary emotional expression. These submantellar areas and tracts, it can also be demonstrated, have structural connections with definite portions of the cortex and especially with frontal areas in which it is universally believed are inhibitory centres or mechanisms, and also, it is believed by some, psycho-excitory centres or mechanisms.

Briefly stated, my views with regard to the cortical representation of emotional expression are that in the midfrontal region of the cerebral hemispheres, and especially on the right side, is an area which may be regarded either as an independent zone or an anterior subdivision of the general motor zone. Here are cortically represented those movements which are especially concerned with the expression of emotion. The term *movement* in this connection is given a broad significance as applying not only to the movements performed by skeletal muscles, as the movements of the face and limbs, but to movements visceral, vascular, secretory, in fact all movements which result in any way in adding to the sum total of emotional expression. Let me say here again that I am not now speaking of areas and centres of emotion, but of emotional expression. That part of the frontal lobe which takes a chief part in the special syntheses which express themselves as the mental process called emotion, is in all probability closely contiguous to this zone of emotional expression. The cortical area of emotional expression, in other words, is intercalated between the motor zone as usually described and the great prefrontal psychic sphere. Perhaps the last interlaces in its structural arrangement with this zone of expressional representation.

This view is not as extreme or as improbable as might to some at first sight appear. The more primitive zone of motor representation in the left hemicerebrum has anterior to it an evolutional outgrowth which corresponds to the cortical motor centre for speech and cortical motor centre for writing. As evolution advances cerebral development takes place in a caudocephalic direction. As the skilled movements of speech and those necessary for writing and drawing have been added to the human equipment, this midfrontal region has more and more attained importance.

Similarly in the cerebral hemispheres, and especially in the right cerebral hemisphere, anterior to the more primitive motor zone has been developed a zone for the representation of emotional expression. Perhaps it may not be spoken of as a region for the representation of skilled movements, or at least this idea is not so apparent as in the case of speech and writing; nevertheless emotional expression correctly regarded holds a higher place than the representation of voluntary movement.

Earlier in this paper I have presented some of my reasons, drawn from faradization of midfrontal and prefrontal cortical areas for regarding this zone of emotional expression as located in front of the motor zone as ordinarily regarded. Some clinicopathological evidence in favor of this hypothesis has also been collected by me, The case, with the smiling or laughing aura, a history of which is appended, being in this category.

In this connection I would call attention to the fact that Nothnagel in discussing his often cited cases of mimetic facial paralysis, so commonly used to uphold the hypothesis of von Bechterew of the thalamic centre for mimetic expression, remarks that further experience may lead to the conclusion that somewhere in the cerebral cortex this emotional innervation may have a definite place, a zone or area which is the rallying point for the psychic reflexes, Impulses arriving by the various tracts of the cortical association systems being gathered here before passing into the tracts through which impulses concerned in emotional expression are discharged. This rendezvous, if it exists, would be the cortical emotive zone suggested by me as located in the midfrontal, or midfrontal and prefrontal cortex.

THE PART PLAYED BY THE THALAMUS AND OTHER BASAL GANGLIA IN THE MECHANISM OF EMOTIONAL EXPRESSION

The thalamus, as has already been indicated more than once in this presentation, has long been supposed to play an important if not a dominant role in emotional expression. Many of the earlier neurophysiologists regarded the thalamus as a motor organ, but even by some of those who had largely disregarded this view of its functions as a whole, some portion of it continued to be looked upon as motor or efferent. Von Bechterew in particular taught that in the thalamus, especially in its medial portion, is a centre for mimetic expression, irritation or destruction of which on one or both sides would cause on the one hand increase in emotional movements or on the other hand paralysis of such movements. This hypothesis of von Bechterew has been adopted by many, and it has seemed to me, as to some others, without adequate experimental or clinicopathological evidence. The main data, physiological and clinicopathological, as given by von Bechterew on which his view is founded, can be summarized about as follows:

(1) If all parts of the brain down to the thalami are removed, the animal loses, along with the psychic functions, its power of voluntary motion; but it retains, along with the simple reflexes, all the movements of expression; among other phenomena many kinds of vocal utterances occur reflexively such as can be evoked in normal animals also. (2) If the cerebral hemispheres together with the thalami are removed the animal loses, along with its psychic functions, the ability to perform all the complex movements of expression; there remain only the simple motor phenomena of a general or local character, like restlessness in pain, as well as reflexive vocal utterances, which in this case are evoked only by the painful irritation, and tend to become more or less monotonous. (3) If one irritate the thalami by penetrating their mass with an electric needle, particularly in their distal part, certain movements of expression are produced, as well as

many kinds of vocal utterances (such as growling, baying, or howling in dogs), which appear the more readily upon bilateral irritation. (4) If one destroys or injures in animals the thalami in their posterior parts, leaving the cerebral hemispheres intact, the power for movements of expression will be more or less modified, along with the remaining motility; indeed, in individual cases it amounts to a mimetic paralysis of the facial muscles while the power of voluntary motion is retained. In the case of a one-sided lesion in the interior of the posterior region of the thalamus, this mimetic facial paralysis with preservation of voluntary movements occurs on the opposite side.

Von Bechterew believes that he has confirmed his experimental investigations and the views based upon them, by personal clinical observations and also by the collection of clinical and clinicopathological observations of others.

On a careful consideration of this exposition of the views of von Bechterew one can readily accept all the facts as given without necessarily concluding that the thalamus is to be regarded as in any of its parts a motor centre. Interference with its functions as a great station on the afferent side of the nervous system might cause just such phenomena as are described by von Bechterew and used by him in upholding his theory of a special motor (mimetic) thalamic centre. Before, however, considering this question from the standpoint of the recent observations and theories regarding the functions of the thalamus, and especially those of Dejerine and Roussy and of Head and Holmes, brief reference might be made to a few of the many neurologists and physiologists of high authority who have supported the stand taken by von Bechterew and also to the views of those who have held that the mimetic centre was placed elsewhere, as in the lenticula.

Nothnagel in discussing his cases of one-sided mimetic facial paralysis adopts the view of von Bechterew with regard to the part played by the thalamus as a psycho-reflex centre for emotional expression. One of his cases, however, because of the size of the lesion which involved much more than the thalamus, does not appear to be conclusive in support of the theory. He clearly shows, however, as others have shown, that in order to explain the phenomena both of emotional paralysis and emotional overaction it is necessary, as regards centres, tracts, and associations, that the mechanism of voluntary movement and that of emotional expression should be largely independent of each other.

Brissaud in one of his instructive and entertaining lectures, in which he discusses involuntary or spasmodic laughing and weeping, seems to adopt the hypothesis of von Bechterew as regards a thalamic mimetic centre, with some special views of his own as to its relations to the cortex and other parts. He shows the evident part played by the bulbar nuclei in emotional expression.

He also shows what is well known, that lesions variously situated as in the bulb, in the posterior or anterior limb of the internal capsule, or in the thalamus may be the pathological cause of the involuntary emotional outbursts. He especially emphasizes the fact that bilateral paralyzing lesions of the voluntary motor bundle in the internal capsule may cause spasmodic laughter or weeping, giving one of the pseudobulbar forms of this affection; and also, he holds that bilateral or perhaps unilateral irritative lesions of the corticothalamic bundles may give the same if the excitation is sufficient.

Brissaud while apparently adopting the views of von Bechterew, at the same time criticises them. He cannot see, as others have not been able to see, how destructive lesions of the thalamus would cause the effect attributed to them by von Bechterew unless the lesions were small and partly of an irritating character.

As indicating the widely separated lesions which may cause involuntary laughing and weeping, and therefore the elaborate extent and distribution of the structural basis of emotional expression, I might refer to the fact that Brissaud cites cases of hemiplegia or hemiparesis, of disseminated sclerosis, of amyotrophic lateral sclerosis, of bilateral primary lateral sclerosis, of apoplectic injury causing paralysis of all four extremities, of diseases due to encephalitis, of simple atrophy of the brain, and of porencephaly.

Brissaud speaks of the important part played by what it seems he first designated, when studying under Charcot, the "psychic bundle," by which is to be understood the fasciculus which passes by way of the anterior limb of the internal capsule from the frontal lobe to the thalamus.

Instead of the thalamus the lenticula has been supposed by some to contain centres for mimetic expression on the efferent side of the nervous mechanism, a view which while not proved has as much in its support as the von Bechterew-Nothnagel thalamic hypothesis. Mingazzini holds that the thalamic fibers probably pass by way of the putamen of the lenticula, believing also that the voluntary geniculate bundle takes part in inhibition. Goldstein appears to maintain a view similar to that of Mingazzini as he refers spasmodic laughing and weeping in some cases at least to lesions of the lenticula.

The weight of recent evidence as well as much that has been learned in the recent or more or less remote past, strengthens the view that the thalamus is an organ entirely on the afferent side of the nervous system. Dejerine and Roussy and Head and Holmes have contributed much to emphasize this position and also to elucidate the functions of the thalamus and the clinical phenomena produced by its disease. All afferent fibers passing upward from the midbrain appear to end in this ganglion, and here, using the language of Head and Holmes, "Lie the synaptic junctions of those paths by which impulses are carried onward to the cortex." I shall not here discuss the thalamic syndrome of the authors just cited, but merely call attention to a few points which seem to bear upon the discussion of the question of the mechanism of emotional expression.

In passing, I might refer to the statement of Malone and Jacobsohn, cited by Lewandowsky, that there is in the thalamus no single cell of motor structure. Lewandowsky's views, as indicated in his discussion of the centripetal, centrifugal, and associating cerebral tracts, are opposed to that of von Bechterew and his followers.

Lewandowsky describes the many connections of the cerebrum with the pontile nuclei, which connections may prove significant in the final elucidation of the question of the efferent side of the emotional motor mechanism.

In a paper like the present, not so much intended for neurologists and neuropathologists as for those generally interested in the question of emotion and emotional expression, it will not be necessary for me to describe in detail the many connections, centripetal and centrifugal, corticofugal and corticopetal, of the thalamus. The work of many neuropathologists, like von Monakow, Obersteiner, Lewandowsky, Holmes, and Spiller, has contributed to our exact knowledge of the elaborate structural relations of the thalamus with other parts — with afferent tegmental tracts, with paths from the cerebellum by way of the nucleus ruber, with to-and-fro connections to all parts of the sensory cerebral cortex, with the frontal region, with the caudatum and with the putamen of the lenticula. What is necessary to be understood is that the thalamus, a great station on the afferent pathway, is so situated as to play a distinctive role in emotion and emotional expression. Painful and pleasureable stimuli are here received, interaction between the sensory cortex and the thalamus taking place in such a way as to bring about a clearer recognition and discrimination of the sensations received, while the frontal cortex acts in an inhibitory or regulating manner on the thalamic centre for sensation, and the cerebellum gives the necessary tone and coördination.

Let me cite here from Head and Holmes an instructive paragraph.

"The anatomical structure known as the optic thalamus is an extremely complex portion of the brain, and contains not only the terminal centre for certain aspects of sensation, but plays a three-fold part in the fate of sensory impulses:

"1. It contains the termination of all secondary paths; here sensory impulses are grouped afresh and redistributed in two directions, on the one hand to the cortex cerebri, and on the other to the gray matter of the optic thalamus itself.

"2. It contains a mass of gray matter, the essential organ of the thalamus, which forms the centre for certain fundamental elements of sensation. It is complementary to the sensory cortex and exercises different functions in the production of sensation.

"3. The lateral part of the optic thalamus is the organ through which the cortex can influence the essential thalamic centre, controlling and checking its activity. The excessive response to affective stimuli, so prominent a feature of lesions in this situation, is not due to irritation but to removal of cortical control."

Excessive response to affective stimuli, painful and pleasurable, is a notable feature in the thalamic syndrome of Roussy as described by him and by Head and Holmes. Some of the cases recorded by these authors have much interest in connection with this subject of emotion and emotional expression and their organic basis. Head and Holmes, for example, speak of two cases in which music produced not only disesthesia or distressing sensations on the side rendered anesthetic by the thalamic lesion, but even choreoid and athetoid movements of the limbs of this side.

EMOTION AND THE RIGHT CEREBRAL HEMISPHERE

I have spoken of the zone of emotion and emotional expression as especially developed in the right cerebral hemisphere. It is not a new idea that the right hemicerebrum plays a larger part in the realization, control, and excitation of emotions than the left. Among those who early discussed this subject was E. C. Seguin, of New York, who believed from his personal observations that emotional expression was most disturbed in cases of right-sided cerebral disease, and, therefore, of course of left-sided paralysis.

Others have contributed now and then to the exposition of this same idea. I have had the matter under observation for many years. Interesting cases of explosive laughter associated with left hemiplegia have fallen under my observation. Usually when involuntary and spasmodic laughter and weeping are the result of a unilateral lesion, that lesion is somewhere in the right half of the brain. It may be a large cortical or subcortical destructive lesion, or it may be, as in one case which I have the opportunity of presenting in this paper through the courtesy of Dr. Weisenburg, a case of lesion of the right ruber and adjoining cerebral peduncle. It may in fact be a lesion anywhere situated in or below the cortex before decussation of the motor pathways.

Emotional representation would seem in some respects to take the place in the right hemisphere which is held by language representation in the left. In the cortex of the right hemisphere are probably more highly developed special centres for the control of the bilateral or median movements concerned chiefly with the expression~~s~~ of emotion.

Both eyebrows, both eyelids, both eyes, both sides of the face, the limbs of both sides take part in emotional expression, and the representation of their movements must, of course, be in correspondence with this fact. Emotion as evidenced by blushing or pallor spreads itself over the entire face or it may be over the entire trunk and limbs. What might be classed as the median or centrally placed movements of expression are really bilateral and represented in each hemisphere, but especially in the right. Such movements are, for example, as has already been discussed, those of the corrugator supercilii and the muscles which act with it in the expression of grief. The movements of opening the mouth might be placed in the same category, while depression of the corners of the mouth may be a unilateral but is most frequently a bilateral movement. Pupillary movements also are of this kind when expressive of emotion.

SOME CASES ILLUSTRATIVE OF DISORDERS OF EMOTIONAL EXPRESSION FROM CEREBRAL DISEASE

My purpose in this contribution has been to present the subject of the mechanism of emotional expression in a general way with lantern slide illustrations to emphasize and add interest. From a large experience I could of course bring many cases of organic nervous disease illustrating special forms and phases of emotional disorder, but this would extend the paper too much. My personal experience includes cases of emotional disorder from pseudobulbar paralysis, from disseminated and other forms of sclerosis, from deep-seated lesions of the pons oblongata, from lesions of the lenticula and of the peduncles, cerebellar and cerebral, and from large or disseminated lesions of the cerebrum itself. These experiences simply confirm the observations of others as summarized when speaking of the theories of the encephalic mechanism of emotion and emotional expression, observations which show how widely separated are the lesions which may cause disruption of this mechanism, and, therefore, how elaborate the structural bases of this mechanism must be. In concluding the paper I shall simply present the details of several hitherto unpublished cases illustrating a few features of the subjects discussed.

CASE I.—*Apoplectic stroke causing left hemiplegia with right third nerve involvement; slight impairment of sensation and decided ataxic tremor on the left; spells of uncontrollable explosive laughter.*

M. M., white, married, a machinist, aged forty-seven years, was admitted to the Philadelphia General Hospital on July 10, 1906, this being his second admission, the first having been in February, 1906.

The following notes were taken just after his admission. He had used alcohol freely. He had a history of chancre twenty years before coming under observation; otherwise he was well until in 1889 he had an apoplectic stroke which paralyzed him in the left arm, leg, and face. The patient said that on the night of the stroke he had a sensation of pins and needles all over the body, and imagined that somebody was playing a practical joke on him with electricity. He arose and looked under the bed for the battery. From this time on he gradually became unconscious and remained so for two days. The stroke confined him to bed for about a year. He had diplopia and right-sided ptosis, which also lasted about one year. The paralysis, both of the left side and of the right third nerve, gradually improved.

The pupils were equal and reacted to light and accommodation. Associated movements were preserved. The nasolabial fold on each side was normal. He had equal control of the muscles on each side of the face. The power of the muscles of mastication was the same on each side. Cutaneous sensation was a little diminished on the left. The grip was not quite so strong on the left as on the right side. No atrophy of the muscles of either arm was present. The triceps and biceps tendon reflexes were more pronounced on the left side than on the right, though they were not much exaggerated.

On the left side the Babinski sign was present, but delayed. The knee-jerks were exaggerated on both sides. Ankle clonus was not obtainable.

The gait was fairly good, though the left leg was dragged slightly. Spasticity was absent.

Notes made in the clinical record on February 10, 1906, state that the history as given above is confirmed. The only remaining sign of his paralysis seemed to be a lessened ability to wink the left eye and a shuffling gait.

The patient stated that after the stroke he laughed almost constantly and uproariously for two years, and that he still did so, but to a much less extent. When he laughs his whole musculature is involved, and tears gather in his eyes. He also seemed to have a coarse, rythmical, to-and-fro tremor of both upper and lower limbs, this being more marked on the left side. On using the finger to nose test the tremor was increased. His memory was good.

Examination in October, 1906, showed no paralysis of the left upper extremity, and some doubtful paresis of the left lower. Ataxia, however, was distinctly present in both left extremities. Sensation was unaffected. The deep and superficial reflexes were increased on the left side and prompt but not exaggerated on the right. Plantar irritation gave extension of the great toe on the left and plantar flexion on the right.

At this time, though not to the same extent as formerly, the patient showed a tendency to break into uncontrollable laughter on a slight or no provocation. He would be talking seriously, and suddenly in the midst of his conversation he would begin to laugh even uproariously. He said he had had this tendency to uncontrollable laughter since his attack in 1889, and that he had never had it before. He said he sometimes made up his mind not to laugh, but it was unavoidable after he had conversed with a person for any length of time.

His laughter was of an explosive and spasmodic character, occurring only at intervals, some of these being long. It was not alternated or intermingled with spells of weeping or howling.

The lesion in this case was probably similar in location to that of the following case in which a necropsy was obtained. In both cases the lesions were right-sided.

For the notes of the following cases I am indebted to Dr. T. H. Weisenburg.

CASE II.—*Apoplectic stroke with left hemiplegia and paralysis of the right third nerve; irrepressible weeping and laughing, chiefly the former; necropsy showing destruction of the right nucleus ruber and partial destruction of the peduncles, cerebral and cerebellar.*

G., a woman, aged fifty-one years. The patient's grandfather and uncle died of paralysis. Her personal history up to the present illness showed normal health. On April 22, 1910, while in the bath-tub, she suddenly fainted and was brought to the Medico-Chirurgical Hospital within forty-eight hours in a semiconscious condition. She was irrational, that is, talked in an incoherent manner; her speech was thick and could not be understood, but she had a tendency to repeat words. At this time the patient had a flaccid paralysis of the left side of the body. The reflexes on the right side were normal. On the left the knee-jerk was absent, but there was present a Babinski reflex. Owing to the patient's mental state the condition as to sensation could not be determined. Ptosis of the right upper lid was present. In smiling only the right side of the face was moved.

In a short time the flaccid condition was succeeded by spasticity, with increase of reflexes, ankle clonus, and Babinski. On the right there was present a complete oculomotor palsy.

From the beginning of her illness the patient was very emotional, crying easily and then again laughing, but crying most often. Occasionally, especially in crying and sometimes in laughing, she would shout loudly. In fact, whenever anyone talked to her she would cry or laugh. Her articulation at first was indistinct, and it was with difficulty that she could be understood; but after a time this was not so marked, and toward the end of her life she could be understood without difficulty. There was never any disturbance in swallowing and chewing.

The patient died four months after the onset of the attack.

Gross examination showed an area of softening in the cerebral peduncle on the right side, destroying entirely the red nucleus, extending into the middle and inner parts of the foot of the peduncle and destroying entirely the intramedullary fibers of the right third nerve.

Microscopically the area of softening was limited to the right nucleus ruber, and the middle portion of the foot of the peduncle (the outer and inner portions being normal), and the intramedullary fibers of the third. The cells of the nuclei in the right third nerve were diminished in number but not diseased. The whole nucleus ruber on the right side was destroyed, the softening extending a little to the left of the median line in a cribriform manner, but not involving the nucleus ruber on the left side. The lesion extended downward into the uppermost part of the pons, its limit being the decussation of the superior cerebellar peduncles. Upward it was limited to the cerebral peduncle alone and did not extend into the thalamus.

CASE III.—*Extensive disease of the right midfrontal and prefrontal region, causing a smiling or laughing aura.*

The following case is worthy of report because of its general clinical and pathological interest, and also in connection with the present contribution because of the peculiar initial bilateral facial spasm or aura which was distinctly of the form of a smile or laugh. The lesions were chiefly in the emotive zone as described in the paper. The case would seem to show at least that the right midfrontal region, and this and a part of the prefrontal cerebral zones are the seat of centres which control the bilateral facial movements of emotional expression. It is to be noted that while most of the time the initial spasm was bilateral and assumed the appearance of a smile or a laugh, at other times it was a unilateral facial movement, this appearing to show that in the diseased area were separate centres for the control of unilateral and bilateral movements of the face.

W. R., aged twenty-nine years, farmer, was first seen in consultation with Dr. M. B. Oberholtzer. After preliminary examination he was admitted to the nervous ward of the hospital of the University of Pennsylvania, where examinations were completed, and the patient was then referred to the surgical service of Dr. Charles H. Frazier for operation. The report of the case as given below has been summarized, omitting some unimportant and negative details of the elaborate examinations which were made.

When an infant the patient had had spasms for a year or two, these ceasing and returning again at intervals of about five years.

Some years ago, probably ten, he began to have occasional peculiar seizures, not very severe in type. In these seizures he had what the doctor, who has seen it more than once, spoke of as a peculiar smile or characteristic smile. It was a smiling expression with a somewhat dazed look, the first appearance being quickly followed by slight or moderate twitchings of the left side of the face. This twitching would often come on as much as a minute after the beginning of the peculiar smile. For several years he had only this sort of spells, in which he did not seem entirely to lose consciousness, although he was somewhat dazed, his consciousness being changed or reduced.

Four years before coming under investigation he began to have much more serious spasmodic or convulsive attacks. These as described by his physician and members of his family were usually preceded by the patient sitting around looking queer, as if something was going to happen. He would then have his so-called characteristic smile, which in about one-half minute or more would be followed by twitching of the left side of the face. This facial spasm or twitching first affected the corner of the mouth, then the cheek in general, next the eyelids, and eventually the entire side of the face. Following this facial spasm, or sometimes during its course, the spasm would extend to the left arm, and in more severe seizures to both arms, but it always predominated on the left side.

Soon after the patient was examined at my office he went with a member of his family and Dr, Edward M. Williams to the University Hospital. While on the way to the hospital he had one of his incomplete seizures. Dr. Williams in reporting his observation of this said: "First was heard a peculiar cackling, high-pitched, explosive laugh. As soon as the face was in view the mouth was seen to be drawn up in both corners and the eyes had a somewhat dull or staring appearance. The patient continued walking during the attack but in a somewhat listless manner, taking short steps, and apparently not knowing where he was going. He was told to stand still until the attack was over. The seizure was not followed by any twitching of the face, arm, or leg. The laughing lasted about one-half minute, and during it there seemed to be some swinging of the arms, which may have been normal, but looked to be in excess of the usual swinging of the arms in walking. As it passed off the patient walked on as usual."

When a child, before he was seven or eight years old, the patient lost considerable weight, and continued to be lacking in this respect. During childhood he had measles, chicken-pox, and at the age of twenty-seven whooping cough. These illnesses did not seem to have any effect upon the spasms.

The patient's head was notably large and unsymmetrical, the right side being considerably larger than the left, especially the frontotemporal region. Inquiry elicited the fact that when the patient was eighteen months old he fell and struck the right side of his head, and after the fall this side became and continued to be more prominent than the left. The patient said that at the age of thirteen, while skating, he fell and again struck the right side of his head, and after this the enlarged side became still larger.

The habits of the patient were good; he did not use tobacco, and alcohol only in very moderate amounts. He denied all venereal disease, and both Wassermann and Noguchi tests made after his admission to the hospital were entirely negative. The family history presented nothing of importance.

The patient was a well-developed man, aged twenty-nine years. He had a good bony framework and his musculature was in good condition. His head was large, of a hydrocephalic type, with a broad, rounded vertex. His hair was black and of the usual amount.

The patient wrinkled his forehead equally and well, and closed his eyelids tightly and equally on both sides. He pouched out his cheeks with somewhat increased expansion on the right side and drew up the left corner of the mouth better than on the right. The masseter muscles were contracted well, possibly a little more forcibly on the right side, which might have been due to the unevenness of his teeth. The tongue protruded in a straight line, there being no tremor. It was free in all directions in its movements. The soft palate moved equally well on both sides.

An eye examination showed that the cornea was clear, conjunctivæ somewhat pale, eyeballs large but not protruding, and the eyebrows very prominent. The eyes moved freely in all directions and there was no disturbance of the visual fields. The pupils were dilated and equal, reacting promptly to light and accommodation. His sight, hearing, and smell were good.

Numerous examinations, general and neurological, showed nothing abnormal except as noted. His lungs, heart, stomach, and viscera generally were examined, and he was also thoroughly investigated for sensation, motility, reflexes, and everything else usually included in a neurological examination. The results were negative.

The following are descriptions of several attacks which the patient had after admission to the hospital:

Dr. Frank D. Reckord, resident physician, made the following note:

"While I was standing but a few feet from the patient he suddenly gave a queer cackle-like laugh. Both sides of the mouth were drawn up and the face wrinkled as if in the act of laughing. This lasted but a few seconds. He then turned his face to the right and buried it in the pillow. At the same time he flexed the right leg on the thigh as if restless. During this time his eyelids were about seven-eighths closed, but at no time did he close them tightly or was there any noticeable tremor. The nose did not quiver—and the attack did not seem to disturb his sleep. In a half minute he again turned his head to the left and kept on sleeping in that position. The attack seemed to be confined to his face."

Other seizures noted were as follows:

At 9 A.M. there was a slight convulsive attack of the muscles of the face and drawing of the mouth to the left. It lasted about a minute, and the patient complained that the jaw was stiff. He had a similar attack at 9 A.M. the next day, and a half hour later he had a laughing aura not followed by convulsion.

Operation was determined upon, and it was planned so as to uncover the midfrontal and prefrontal region, that is, those parts of the frontal lobe cephalad of the precentral convolution. It was decided upon with the hope of either finding a diseased mass which could be removed or treated, or failing this, of using cortical excision.

The skull was opened on the right side by Dr. C. H. Frazier so as to expose the brain anterior to the Rolandic fissure, including a portion of the first, second, and third frontal convolutions. The appearance of the dura was abnormal, being somewhat cloudy and cystic looking. On cutting the dura there was a gush of fluid and an almost immediate collapse of the dura. The dura was seen to be thickened and adherent to the pia-arachnoid, and these in turn to the brain substance underneath. When the membranes were incised and lifted as far as possible out of the field of operation, a strange looking mass was revealed, consisting of fluid mingled with a fatty, gelatinoid, stringy, irregular mass of tissue, resembling in no way brain substance. The cyst, if it could be so called, was divided by strands or portions of this irregular mass so as to give the whole a somewhat multilocular appearance. The diseased mass, as above imperfectly described, included every portion of the exposed area. This exposure in accordance with the measurements and our observation extended from the anterior boundary of the precentral convolution almost to the anterior pole of the brain and from a superior level about an inch from the mesal line about the Sylvian fissure below.

Small specimens removed from the diseased mass were examined in the laboratory of surgical pathology and the following report was made:

The specimen consisted of two small sections of friable tissue, whitish in color, about 1 cm. in diameter.

On microscopic examination a diseased condition was seen indicating a pathological process of some sort which had resulted in atrophy and other destructive tissue changes, so that little if any normal structure was apparent. Here and there a few large cells could be seen in a partial state of preservation, and there were many clear spaces in the tissue, due evidently to some dilatation of the bloodvessels. There was no evidence of tumor formation or of any specific inflammation.

LANTERN SLIDE ILLUSTRATIONS

The following lantern slides were used in illustration of points brought out in the paper:

No. 1. (*a*) Normal appearance of the face in a case of paralysis of emotional expression on the right side; (*b*) voluntary movement of the face on the side of the paralysis of emotional expression.

No. 2. (*a*) Bilateral voluntary movement of the face in showing the teeth; (*b*) paralysis of emotional expression on the right side of the face shown in laughing.

No. 3. (*a*) Partial one-sided voluntary paralysis shown in bilateral movement of the face; (*b*) decided emotional paralysis as shown in laughing (Spiller).

No. 4. Head of snarling dog (Darwin after Wood).

No. 5. Face of dog dissected (Bell).

No. 6. (*a*) Face of horse; (*b*) face of horse dissected (Bell).

No. 7. Chimpanzee, " Peter," showing anxiety and uncertainty (Witmer).

No. 8. Muscles of the human face (Darwin after Bell and Henle).

No. 9. (*a*) Face in laughter; (*b*) muscles used in laughing (McClellan).

No. 10. (*a*) Face showing grief; (*b*) muscles used in expressing grief (McClellan).

No. 11. Scheme of the motor and motor-emotive zones.

No. 12. (*a*) Actor imitating crying; (*b*) imitating care (Borée.)

No. 13. (*a*) Actor imitating *ha, ha* laughing; (*b*) imitating forced, half-hearted laughing (Borée).

No. 14. (*a*) Child spontaneously weeping; (*b*) child spontaneously laughing (Darwin).

No. 15. Gwynplaine (from L'Homme qui Rit, 1869, illustrated by Vierge).

No. 16. Permanent grin, from bilateral brain lesion.

No. 17. (*a*) Normal appearance of face; (*b*) beginning to laugh; (*c*) laughing (Brissaud).

No. 18. (*a*) Usual appearance of face; (*b*) laughing; (*c*) weeping, in pseudobulbar paralysis.

No. 19. Violent weeping with lacrymation in cerebro-bulbo-cerebellar sclerosis (Weisenburg).

No. 20. Expression of anguish with howling, in bilateral brain lesion.

No. 21. (*a*) Expression of grief with forced attitude, standing; (*b*) painful expression, forced attitude, sitting.

No. 22. French Assembly taking the oath of allegiance to the new Republic (McClellan after David).

No. 23. Cartoon of nude figures illustrating muscular action in David's picture (McClellan after David).

No. 24. Bilateral lesions of the lenticula and internal capsule (Lloyd).

No. 25. Lesions of the pons.

No. 26. Lesion of the dentate nucleus, cerebellar cortex, and superior cerebellar peduncle.

No. 27. (*a*) Scheme of the mechanism of emotional expression, after Obersteiner; (*b*) main subdivisions of internal capsule (Obersteiner).

No. 28. Sensory or afferent emotional mechanism.

No. 29. Motor or efferent emotional mechanism.

No. 30. New scheme of efferent and re-enforcing mechanism.

Nos. 31, 32, and 33. Slide showing eyes interchanged in faces of man and woman (Brunton).

Bibliography

Bechterew, W. von. Experimentelle Untersuchungen über Ausdrucksbewegungen bei Tieren. (Experimental Investigations on the Movements of Expression in Animals.) Wratsch (Russia), 1883.

Bechterew, W. von. Unaufhaltsame Lachen und Weinen bei Hirnaffektionen. (Uncontrollable Laughing and Weeping in Affections of the Brain.) Arch. f. Psychiatrie und Nervenkrankheiten, 1894, Band xxvi.

Bechterew, W. von. Ueber die sensible und motorische Rolle des Sehhügels, Monatsschrift für Psychiatrie und Neurologie, Band xvii, Heft 3, S. 224–231.

Bell, Sir Charles. Essays on the Anatomy of Expression in Painting. London: Printed (by C. Wittingham) for Longman, Hurst, Rees, and Orme, Paternoster-row, 1806.

Bianchi, Leonardo. A Text-book of Psychiatry for Physicians and Students. Authorized translation from the Italian by James H. Macdonald, M.D., Ch.B., Glasgow. New York: William Wood & Company, 1906.

Borée, Albert. Physiognomische Studien. 119 Autotypien nach Naturaufnahmen nebst einem kurzen erläuternden Text. Stuttgart: Verlag von Julius Hoffmann, 1899.

Brissaud, E. Leçons sur les Maladies nerveuses. (Salpêtrière, 1893–1894.) Receuillies et publiées par Henri Meige. Paris: G. Masson, Libraire de l'Academie de Medicine, 1895.

Calkins, Mary Whiton. A First Book in Psychology. New York: The Macmillan Company, 1910.

Darwin, Charles. The Expression of the Emotions in Man and Animals. London: John Murray, 1872.

Dejerine et Roussy. Le Syndrome thalamique. Rev. neurol., 1906, tome xiv, p. 521.

Deroubaix. Journal de Neurologie, 1906, Tome ii, No. 5.

Goldstein. Contributioni la studini paraliziei pseudo-bulbaire. (Contributions to the Study of Pseudo-bulbar Paralysis.) Bukarest, 1901. (Cited by Deroubaix.)

Head, Henry, and Holmes, Gordon. Sensory Disturbances from Cerebral Lesions. Brain, 1911, xxxiv, 102.

Holmes, Gordon, and Head, Henry. A Case of Lesion of the Optic Thalamus with Autopsy, Brain, 1911, xxxiv, 255.

Hugo, Victor. The Man Who Laughs. Works, Vol. 12. Boston: Jenson Society, 1900.

Irons, D. The Nature of Emotion. Philosophical Review, vol. vi, 1897, pages 242–256, 471–496.

James, William. Psychology. New York: Henry Holt & Company, 1893.

Lavater, J. C. Essai sur la physiognomie, destineé à faire connaitre l'homme et à le faire aimer.

Lewandowsky, M. Systematisch anatomische Vorbemerkungen über die cerebrofugalen Leitungsbahnen (Systematic Anatomical Introductory Remarks on the Cerebrofugal Conduction Tracts). In Lewandowsky's Handbuch der Neurologie, erster band, allgemeine Neurologie, ii, Berlin, 1910.

Lloyd, James Hendrie. Pseudo-bulbar Palsy. Philadelphia General Hospital Reports, vol. vii, p. 142.

McClellan, George. Anatomy in its Relation to Art. Philadelphia: Printed for the author by A. M. Slocum & Company, 1900.

Mantegazza, Paolo. Physiognomy and Expression. London: Walter Scott, 1892.

Mills, Charles K. The Nervous System and its Diseases. Philadelphia: Lippincott, 1898.

Mills, Charles K. Preliminary Note on a New Symptom Complex Due to Lesion of the Cerebellum and Cerebello-rubro-thalamic System, etc. The Journal of Nervous and Mental Disease, February, 1912, vol. xxxix, No. 2.

Mills, Charles K., and Frazier, Charles H. The Motor Area of the Human Cerebrum, its Position and Subdivisions, with Some Discussion of the Surgery of this Area. The University of Pennsylvania Medical Bulletin, July and August, 1905.

Mills, Charles K., and Spiller, William G. The Symptomatology of Lesions of the Lenticular Zone with Some Discussion of the Pathology of Aphasia. The Journal of Nervous and Mental Disease, August and September, 1907.

Mingazzini, Giovanni. Sulla Sintomatologia delle lesioni del nucleo lenticulare. (On the Symptomatology of the Lesions of the Lenticular Nucleus.) Rivista Sperimentale di Freniatria, vol. xxviii, fasc. II–III.

Monakow, C. von. Gehirnpathologie. Nothnagel's Specielle Pathologie und Therapie, Band xi, I Theil. Wien: Hölder, 1897.

Nothnagel, Hermann. Topische Diagnostik der Gehirnkrankheiten, Berlin, 1879.

Nothnagel, Hermann. Zur Diagnose der Sehhügelkrankungen. (Diagnosis of Thalamic Diseases.) Zeitschrift für klinische Medizin, 1889, Band xvi.

Obersteiner, Heinrich. Anleitung beim Studium des Baues nervösen Zentralorgane im gesunden und kranken Zustande, 5te Auf. Leipzig und Wien: Deuticke, 1912.

Oppenheim, H., und Siemerling, E. Mittheilung über Pseudobulbär-paralyse und akute Bulbärparalyse. (Contribution on Pseudobulbar Paralysis and Acute Bulbar Paralysis.) Berliner klinische Wochenschrift, November 15, 1886, no. 46, p. 791.

Spiller, William G. American Journal of the Medical Sciences, March, 1912, vol. cxliii, No. 3, page 390.

Spiller, William G., and Camp, Carl D. The Sensory Tract in Relation to the Inner Capsule. The Journal of Nervous and Mental Disease, 1911.

Titchener, Edward Bradford. A Text-book of Psychology. New York: The Macmillan Company, 1910.

Weisenburg, T. H. Pseudobulbar Palsy. University of Pennsylvania Medical Bulletin, January, 1905.

Weisenburg, T. H., and Guilfoyle, W. F. Tumor Destroying Most of One Optic Thalamus and Causing Central Pains, etc. Review of Neurology and Psychiatry, June, 1910.

Weisenburg, T. H., and Ingham, S. D. Multiple Sclerosis with Primary Degeneration of the Motor Columns and Hypoplasia, Principally of the Brain Stem. The Journal of Nervous and Mental Disease, November, 1910, vol. xxxvii, no. 11.

Wundt, Wilhelm Max. Grundzuege des physiologischen Psychologiche, 5te Auf., Band ii, Leipzig: Engelmann, 1902–1903.

Reprinted from the Transactions of the College of Physicians of Philadelphia, 1912

DISCUSSION OF DR. MILLS' PAPER ON THE CEREBRAL MECHANISM OF EMOTIONAL EXPRESSION

DR. GEORGE MCCLELLAN: Who of us all can speak so authoritatively on such a subtle subject as Dr. Mills?

As for venturing to question his conclusions I will not be so bold, especially in the companionship of those whom he has chosen to speak with me on *the mechanism of emotional expression.*

It would be difficult, indeed, to add to the interest of Dr. Mills' lecture, but in the brief time allotted to me I will try to accentuate certain points to which I have given special attention; after first referring to one of the convictions of a man of great ability with whose writings you are all more or less acquainted. There are few here to-night who do not know Sir Lauder Brunton. I was surprised, one of the last times I saw him in his own house, when he referred to his belief that very little, if any, expression could be attributed to *the eyes themselves,* and that all seeming variety of emotion betrayed by them was due to their more or less immediate surroundings.

In illustration, he presented me with a couple of slides, which he had been in the habit of using at the time he taught the anatomy of expression in his lectures at Edinburgh. They are ingeniously conceived. On one plate a pair of eyes, pure and simple, entirely without eyelids, lashes, or facial surroundings; on another glass slide are the face of a drunkard and that of a nun, the latter with brow uplifted as in prayer. The eyes of each face are omitted, but so arranged that they can be superimposed upon the first plate and the eyeballs seen in these varying settings, which certainly alter their whole effect and import to a startling degree. (I will show these to you presently). While admitting the value of his observations, I cannot entirely indorse the conclusion of Dr. Brunton.

It is certainly evident that the human countenance betrays changes of expression more through the surrounding eye accessories, the brows, the lids, etc., than by the eyeballs themselves, yet there are times when in the recognition of the emotions especially one is convinced that the eyes are the "*windows of the soul.*"

It is the muscles about the eyes, under different influences, which convey the typical expressions in *laughter*, *astonishment*, *horror*, *anger*, *disgust*, or *grief;* but it is not easy to believe that the distinction of these expressions could be conveyed by a series of glass eyes, however artfully managed or surrounded. Surely the light of merriment, the profundity of grief, the glare of hatred or of horror, are not mere optical illusions of our imperfect vision, any more than the sudden flash of surprise or delight could be stimulated by a doll or a puppet with the cleverest mechanism.

How without the eyes can be interpreted the strength, the abandonment, or the intensity of these expressions, which are all produced by the same facial muscles with varying contractions? Surely each would lose all vital force and meaning if the eyes themselves did not play a leading part in them. The artist tries to illustrate the vital spark when he indicates the effect of the light upon the conjunctiva. Photography sometimes reproduces the high lights, but too often with a set expression in the muscles about the eye. (I will show you some slides of my drawings taken from my book *Anatomy in its Relation to Art*, which were suggested by a remarkable work entitled *Symphonies d'Expression*, by Maurice Heyman.)

The subtle distinction made by Dr. Mills between histrionic expression, whether caused by simulated or genuine emotion, and the undoubted effect of spontaneous feeling, in the expressions of the human face leads me to explain that these studies of emotional display, by Heyman, belong more properly to the former class.

As is well known (to anatomists) the muscles of expression are all of finer texture than the muscles generally in the other parts of the body. These muscles have no proper tendons of origin or insertion, are destitute of sheaths and blend with adjacent muscles by the most complex and delicate interlacement of their fibers, so that they do not present the same arrangement in every individual. They even vary in development on the sides of the same face, sometimes thereby influencing the character or degree of the expression. (This is curiously conspicuous in the statue of Voltaire at the Theatre Français, Paris, and of Blackstone in the Bodlein Library at Oxford.)

In seventeen consecutive dissections of the face I have found no two faces with equal development of the muscles on both sides. The chief deviation had been in the zygomatic muscles and those about the angles of the mouth. The intimate association of the skin with many of the adjacent muscles produces through their habitual use the facial dimples, furrows, and markings, characteristic of certain expressions. This is especially noticeable in young children.

In the ignorant who cannot readily communicate ideas or describe sensations by speech, these lines are most valuable as indications, but they are usually very much exaggerated. It should be remembered that the changes of the features in the human countenance, where they are not due to the influence of the mind, but are expressions characteristic of strong emotions, passions, and affections, are the physical manifestations of impressions upon *the heart and lungs*. So are also the contortions of a child's face at birth, in this case almost automatic, before the brain is capable of mental effort.

The habitual expressions which stamp the features with individual character are often due to the exercise of the will in self-control, or to brooding of the mind upon certain passions.

Reverting to the eyes and the muscles which act upon them, elevation of character is often indicated by lines upon the forehead, which result from the habitual play of the muscles surrounding the eye and the muscles of its orbit.

Sir Charles Bell attributes to the eye the loftier emotions and describes the effect of its influence in reverence, in devotion, in agony of mind, in all sentiments of pity. The effect of power is greatly due to the projecting eyebrow. Beauty usually implies a large eye, and it is in the eye that one looks for sentiment and reproof.

The straight muscles of the eyeball are governed by the will alone, but the oblique muscles have sympathetic filaments which bring them under the influence of the sympathetic nervous system, so that they act when the other muscles cease to operate. Hence in sleep, in fainting, or in approaching death the eyeballs are revolved. In forced inspiration we instinctively droop the lids, and in sneezing and coughing it is the same.

The group of muscles of the upper part of the human face which by their action produce the so-called *intellectual expressions* give character to the features according to the predominant traits of the individual, even in repose, or after death. Bell pointed out that their function is to produce the *nobler* expressions of the human countenance, in contrast to that of the muscles peculiarly *animal* which act upon the lower part of the face.

Careful study of the lines of the face and their varying modifications prove of great value to the physician in forming a diagnosis of many diseases, especially in young children, and the ignorant, in whom the facial expression is often the sole guide to the recognition of subjective symptoms. The only way of obtaining information from an infant, who cannot communicate ideas or describe sensations by speech, is by noticing the expressions and gestures.

It should be noted that the expression of the emotions is not confined alone to the countenance, but that coincidentally there are movements of the rest of the body with characteristic postures and contortions, which participate in conveying the meaning. This is particularly the case in the self-abandonment of the savage among all races; yet it is a curious contradiction in fact, that while self-control is undoubtedly one of the acquirements of civilization, no statesman nor diplomatist can boast a more absolutely non-committal countenance and demeanor than is presented by the North American Indian, when such is his will.

That wonderful nerve, the vagus, is chiefly concerned in expression, through its connection with the larynx, the heart, the lungs, and the stomach. Of course, too, the facial and auditory nerves play conspicuous parts, and it is to be emphasized that the origins of all these nerves are very close to one another—mostly in the floor of the fourth ventricle (of the cerebellum), and it is not difficult to conjecture that nerves of communication may be established with certain areas of the cerebral cortex, especially with the frontal and central convolutions, and the cuneate lobes, as Dr. Mills has so clearly and ably pointed out.

Dr. James Hendrie Lloyd: The subject is widely speculative, but I think it is a great deal better that we confine ourselves to what we have learned at the bedside and the autopsy table. Dr. Mills has given us some illustrations of automatic or bulbar laughter, one of which was taken from a case of my own. We have in these cases a paralysis of certain muscles of the face, throat, neck, tongue, and sometimes of the eyes, caused by lesions above the nuclei of the third, fifth, seventh, tenth, and twelfth nerves. The lesions are frequently in the lenticular nucleus, cutting off the nuclei of these particular nerves in the midbrain, pons, and medulla from the cortical centres in the brain. It is in these cases particularly, called pseudobulbar palsy, that we have automatic laughter, in which the patient is seized with paroxysms of uncontrollable laughter or sometimes of weeping. It is a misnomer to call it, as some have done, epileptic laughter, for there is nothing epileptic about it. Dr. Mills has exhibited illustrations of the microscopic sections in my own case. These accord with some few of the other cases reported in the literature. The lesions are in the lenticular nuclei on both sides and must cut off the corticobulbar fibers, *i. e.*, the fibers running from the motor centres in the cortex of the brain to the nuclei of the cranial nerves in the pons and medulla. There are a great many speculative theories about this phenomenon of automatic laughter. In the first place, if you come to think about it, this whole subject of laughter and weeping is curious. Why do we laugh or weep? There is no necessary or logical connection between the emotional condition in the mind and these peculiar spasmodic motions of the face which we call laughter and weeping. This function is confined to the human race, for the lower animals neither laugh nor weep. Since the days when our ancestors were apes we have in some way by long practice and habit evolved this peculiar function called laughter and weeping. My own theory is not so elaborate as that of others. My explanation is that we have evolved a sort of coördinated automatic apparatus which goes off practically almost by itself. It is like other automatic or semi-automatic functions, such as walking, talking, and writing, and can best be illustrated by an example. As a student I remember that Professor Gurney Smith used to show us a decapitated frog. When there was put upon the frog's leg a little drop of acid the creature would try with the foot of the other leg to scrape it off. Something like it is seen also in the decapitated rattlesnake: if its tail is trodden on the creature rears its headless trunk and strikes. This looks like a volitional act, but it is purely automatic. I do not think it is necessary to make up an elaborate series of explanations and schemata, which, indeed, to me are rather confusing. I believe the simplest explanation is the best; and in these cases (just as in the decapitated frog) the lesions in the lenticular nuclei act to cut off the volitional centres in the brain from the mechanical centres in the bulb.

Dr. Smith Ely Jelliffe: It gives me great pleasure to be here and especially to have heard the delightful address that Dr. Mills has given us, to have seen the beautiful photographs and to have learned so much from those who have preceded me in the discussion.

It seems to me, for myself at least, there is very little to add to the discussion, and yet I cannot refrain from saying just a few words that have suggested themselves to me while listening to Dr. Mills and seeing Dr. Weisenburg's splendid cinematograph demonstrations. I have prepared no elaborate discussion, and only wish, as it were, to give my own psychosensory reflexes as a result of the stimuli that I have received in the preceding hour.

This is a very old subject. Homer devoted considerable space to the question of sadness and of joy. Pliny said that the centre for laughter was in the diaphragm. An early Latin poet said, "Beware of the laughter that shows itself only in the mouth." So that it has been recognized from time immemorial that the motor manifestations underlying two very primary principles in life—pleasure and pain—so far as emotional expression is concerned, show very complicated mechanisms. The mechanisms of emotional expression, are not as simple as Dr. Lloyd would have us believe, nor do the demonstrations we have seen permit us to dispose of the matter by the simple scheme suggested by Dr. Lloyd. If man were a simple spinobulbar animal, then the elementary facts touched upon by Dr. Lloyd would be sufficient, but fortunately we are not frogs. Human evolution has permitted the development of various cortical and subcortical structures. In order to excite these new psycho-motor-sensory reflexes an extremely complex mechanism has developed.

In the first place, I have heard nothing of the influences of sensory stimuli coming through the trigeminus. This sensory distribution of the face gives us all our information about the way the muscles of our face are set. Its functional integrity determines the postural muscular adaptations as well as the efficacy of the willed movements and the psychoreflex movements. The trigeminus is only one of the important factors in the parallelogram of forces. We must think of the subject of emotional expression as a psycho-sensory-motor reflex. I believe we have a vast polygon of forces to be analyzed from the sensory ~~motor~~ as well as from the motor side. Thus in pseudobulbar paralysis there is no emotion with the laughter which we see. Here there is a modified reflex action, not an emotional disturbance. The subjects do not feel happy. They do not feel sad.

I am in accord with Dr. Mills in assuming that Bechterew's view will not hold. The thalamus is an important station for the reception of practically all of those sensations which underlie our affective reactions. In this sense it is a centre, not for emotional expression, but for perceptions, its disorders influence emotional expression but they are not responsible for the impulsive laughter and crying mimicry movements. These we shall find in a number of places in the cortex, in the midbrain, in the pons, medulla, cranial nerves, and in the spinal cord. All contribute elements to that polygon of forces, the disturbances of which result in the modifications of emotional expression so well illustrated this evening.

Dr. William G. Spiller: I know of no paper in which this subject has been treated so comprehensively as in the paper Dr. Mills has presented this evening. I also know of no case in which the paralysis of emotional movement has been so distinct as in Dr. Mills' patient. I have been interested in observing that in a number of persons the movements of the face in emotional expression are deficient on one side. It is rare that this leads to any misconception as to paralysis, yet last year a patient was brought to me in whom facial paralysis was feared, because of imperfect movement of one side of the face in smiling. I have never seen bilateral deficiency of emotional movement.

Probst discovered that after destruction of the cerebellum there is impairment of muscular sense and of the sense of position, from which the conclusion may be drawn that a part of the sensory conduction is through the cerebellum. The loss of emotional movement, which movement is largely a reflex, may depend on the loss of these sensory fibers passing through the cerebellum. They may have their course in the fifth or seventh nerve, and I believe they are represented in the latter. Head has shown that the deep sensation is dependent on the nerves to the muscles, and I believe such fibers probably exist in the seventh nerve. Sherrington has demonstrated that the ocular nerves contain afferent fibers, and the function of these must be for muscular sense. I doubt whether any nerve has purely efferent fibers.

Dr. Augustus A. Eshner: Naturally there are a number of phases to be taken into consideration in a discussion of emotional expression. Dr. Mills has dwelt most largely upon the muscular expression of this phenomenon. Naturally no little consideration must be given to the exciting factors, and in connection with these the individual susceptibility to such stimuli also must be taken into account. As is perfectly well known, of any group of individuals exposed to a given psychic stimulus there will be varying degrees of response in the way of emotional expression, whether of sadness or of joy respectively.

There is another point to which I shall allude and to which Dr. Jelliffe has called attention, namely that in cases of bulbar paralysis and multiple sclerosis the muscular expression of emotion is not necessarily indicative of any affective disturbance, but represents merely muscular acts in response to influences beyond our immediate observation, and they may be considered, it seems to me, as rather counterfeits of emotional expression.

So, too, with the histrionic reproductions of emotional states. These, I take it, are mere superficial imitations, and naturally cannot be considered as expressive of deep-seated emotions. I doubt that any individual, however clever his histrionic ability, is capable, beyond a certain limited degree, of reproducing a true emotion, except in this superficial form, which must in the final analysis be considered merely as a simulation or as a counterfeit.

Dr. J. Cardeen Cooper: I did not hear Dr. Mills' paper, but there are two points in the discussion to which I should like to refer. One was made by Dr. McClellan in saying that the blank and sullen expression of the face is only noticeable in man, and is the result of education and civilization; and yet he refers to the North American Indian as showing often a marked degree of this tranquillity. It was said of Napoleon I that he had this ability of expression to a greater degree than any other man of his time. Carlisle in his *History of the French Revolution* said that this was not due to education, but rather to his supreme will; and I have read somewhere that this absence of emotional expression is true of the North American Indian. It would seem, therefore, that the person did not have to be educated in order to dismiss from his face all lines of facial emotion and expression.

I wish to call attention to one point made in Dr. Lloyd's discussion, "that man is the only animal that shows the emotion and expression of laughter or grief." Dr. Pleuvences, of Paris, has said that Darwin in his *Descent of Man* says that all animals, such as cattle, dogs, and horses, show the very same expressions of laughter, grief, and various emotions as does man, and that the difficulty is in the inability of the observer to detect these expressions in the particular facial display of the muscles. Attention has also been called to the fact that a retriever, invariably, when a bird has been wounded or killed and brought to its master, would show the most decided expression of laughter in his face. Dogs also show a most decided expression of grief when conditions pertaining to grief are present.

Dr. Mills, closing: The College has already been liberal in the time allowed for the paper and the discussion. I will say only a word in regard to a remark made by Dr. Jelliffe in reference to the part played by sensation. The subject is so large that I gave only the merest outlines of what I had to present. If I had had time to demonstrate the three schemes of the mechanism of emotional expression which were thrown on the screen, he would have seen that one was illustrative of the sensory or afferent side of this mechanism, and the importance accorded to sensory pathways and centres would have been more evident.

Reprinted from Brain, Vol. XXXVI, pp. 75-114, *July*, 1913.

THE RELATION OF THE MYOPATHIES.[1]

BY WILLIAM G. SPILLER, M.D.

Professor of Neuropathology in the University of Pennsylvania, Philadelphia.

The classification of the myopathies is no easy task, but we may recognize two great groups of muscular atrophy or arrest, (1) the congenital, (2) the acquired.

The congenital embraces the cases of arrested growth in certain limited regions of the body, the infantile nuclear arrest ("Kernschwund") of Möbius. This muscular condition may be caused by defect of muscles with complete integrity of the nerve apparatus, or, on the other hand, the peripheral neurones may be much affected. The congenital arrest usually is not progressive, although it may be progressive in exceptional cases.

The acquired muscular atrophy also consists of two types, the primarily muscular, with intact nervous system, and the nuclear (neuronic), in which the nerve-cells of the peripheral neurones are affected. In the former we have the progressive muscular dystrophy, including the bulbar form of Hoffmann; in the latter we have the Werdnig-Hoffmann muscular atrophy, the infantile bulbar atrophy of Fazio, Charcot, and Londe, and the forms of myelopathy occurring later than the period of childhood. These types, as is well known, are progressive.

The various forms are combined in rare cases, and certain of the muscles of the body, as the pectorals and serratus magnus, show a special liability to arrest or atrophy in both forms. It is also well known that reaction of degeneration and fibrillary tremors, as well as changes in the cells of the anterior horns, have been observed in progressive muscular dystrophy, so that sharp distinctions cannot invariably be maintained.

The relation of muscular defect to progressive muscular dystrophy has been recognized by a number of investigators. By some the defect has been considered as arrested intra-uterine dystrophy. In persisting portions of arrested muscles the histological findings have been those suggesting intra-uterine muscular dystrophy, and yet one must be cautious in accepting these findings as conclusive, for it is questionable whether a decision can be formed as to the myopathic or myelopathic character of atrophy from the microscopical findings in the muscles alone. In a paper published in 1898, and in another published in 1900, I called attention to the fact, even at that time well known, that the distinctions which had been sharply drawn between "degenerative" and "simple" muscular atrophy were not trustworthy, and that the histological investigation of the muscles could not determine the myelopathic or myopathic nature of the atrophy [52]. Combinations of muscular defect with muscular dystrophy have been described.

---

[1] This paper was laid before the Neuropathological Section of the International Medical Congress [August 9, 1913].

Recently we have had the classification of myopathy considered in an able article by Frederick E. Batten [3]. His classification affords an excellent basis for discussion. It is as follows:—

(1) The simple atrophic type (myatonia congenita or amyotonia congenita).

(2) The pseudo-hypertrophic type.

(3) The juvenile type (Erb).

(4) The facio-scapulo-humeral type (Landouzy and Dejerine).

(5) The distal type (Gowers).

(6) The myotonia atrophica type.

(7) Mixed and transitional types.

To these I add a somewhat questionable type:—

(8) The true hypertrophic type.

I shall have little to say regarding the types mentioned in the headings 2, 3 and 4; they have been the subject of innumerable papers by Duchenne, Erb, Landouzy and Dejerine, and a host of others. Their sharp limitation, however, is still a matter of dispute, and among recent writers on this subject may be mentioned Eulenburg and Toby Cohn [25]. These authors show by the report of five cases of dystrophy in one family that it may be impossible to class a given case under any one of the recognized types. They maintain that the types are useful for didactic purposes, but should not be too sharply insisted on, and with this view we must agree.

The simple atrophic type, as described by Batten, is one of the most disputed. It is congenital or begins in early infancy, and is characterized by smallness, lack of power and loss of tone in all the muscles of the body, without localized atrophy or hypertrophy of individual muscles or groups of muscles. All movements are performed in a feeble manner. The disease is slowly progressive, and, as a rule, walking is never learnt. The muscles are very flaccid, and the hands and feet can be bent at unusual angles to the arms and legs. Some contractions of the flexors take place late in the disease. With this type Batten assimilates amyotonia congenita, and unquestionably there are many points of resemblance. The distinctions, as Collier and Wilson [14] formulate them, are:—

(1) The myopathies are conspicuously familial diseases, whereas no familial tendency has been recorded in amyotonia.

(2) The several types of myopathy often show familial relationship one with another, whereas no case of amyotonia has been reported in the myopathic family.

(3) A large majority of the cases of amyotonia are congenital, the condition being obvious at birth. In a minority of cases amyotonia has appeared acutely and has reached its most severe degree in a few days. In none of its several types is myopathy present at birth, nor does it ever appear acutely or reach its maximum in a few days.

(4) The characteristic muscular flaccidity is not present in myopathy.

(5) The local muscular wasting that is a marked feature of myopathy is not present in amyotonia.

(6) The course of myopathy is one of progressive muscular weakness, that of amyotonia is one of progressive amelioration of the symptoms.

(7) Return of the deep reflexes after their persistent absence for months or years has been recorded several times in amyotonia, and has occurred in two of the cases reported by Collier and Wilson. Such a return of an absent deep reflex has never been recorded in myopathy.

To these distinctions we may add two presented by Collier and Holmes, viz.:—

(8) Affection of the periphery of the limbs, and especially of the intrinsic hand muscles, which is the invariable rule in amyotonia, is of the greatest rarity in any form of myopathy.

In comparison with this positive statement, Archangelsky and Abrikosoff assert that the feet and hands in amyotonia usually are affected in less degree than the central portions of the limbs.

(9) Amyotonia never spreads to regions previously unaffected. Slow spreading of the affection from muscle to muscle is characteristic of all forms of myopathy.

Batten attempts to show that amyotonia may be a family disease, and in support of this argument he refers to the two cases of amyotonia in children of one family reported by Sylvestri. He refers also to the three cases in one family recorded by Finkelnburg, in one of which a pathological examination was obtained. He adds that one of the children, D.S., recorded by himself as having the simple atrophic type, had a younger brother similarly affected, although at the time of publication he was ignorant of this. These facts, Batten thinks, dispose of the first two points raised, viz., they demonstrate that amyotonia may be a familial disease and may occur in a myopathic family.

Cassirer [11] does not regard the cases of Finkelnburg in discussing the familial occurrence of amyotonia congenita, and states that only Sorgente and Beevor have described cases occurring in the same family, and these, he thinks, are questionable cases. He refers, however, to Sylvestri's cases which presented the characteristic symptoms of the disease, but regards them as uncertain. Further in his article Cassirer speaks of the familial form as described by Collier and Wilson, but regards all cases as "nicht ganz einwandfrei." It may be well to make a brief reference to these cases here.

Batten gives Sylvestri's cases as follows: S. recorded two cases of amyotonia in one family, the first and fifth children; the second and third being healthy, the fourth being a cretin. A maternal aunt suffered from the Landouzy-Dejerine form of myopathy. The elder child, at the age of 16, is said to have presented a typical picture of Erb's juvenile form. Sylvestri's description of the later development of this child is somewhat obscure.

Sevestre's [51] cases: A child, aged 2½ months, had rom birth flaccid paralysis with atrophy of the four limbs and trunk, except the diaphragm. Two other children in the family had a similar disorder. The paralysis was almost complete.

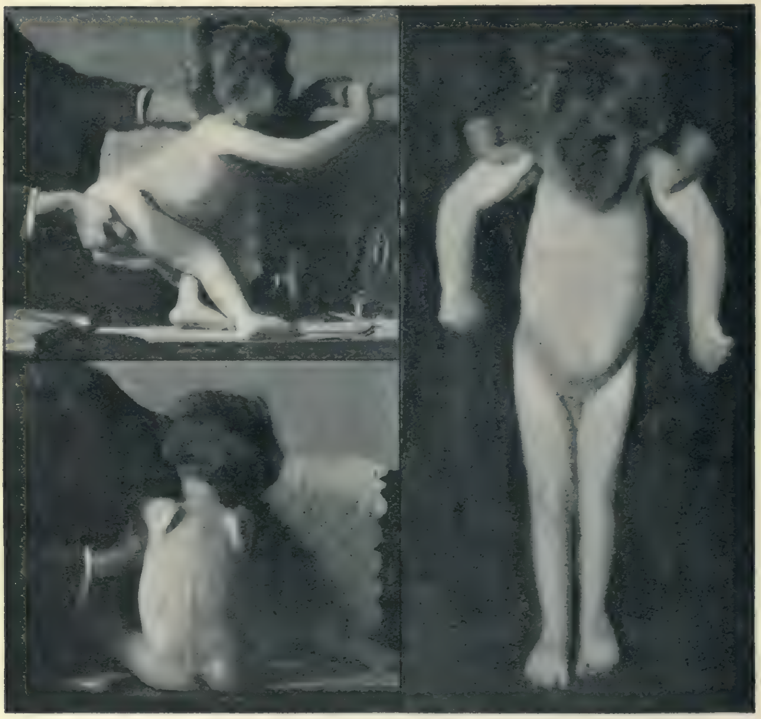
The cases of Sorgente, brother and sister, as given in abstract by Collier and Wilson, presented the symptoms of amyotonia, and apparently nothing contrary to the complex, unless possibly that there was no reaction in muscles of the legs to either faradic or galvanic current, and convulsions occurred before death. These were familial cases, and were without necropsy.

The case that Beevor described in BRAIN, 1902, was regarded by him as belonging to the Werdnig-Hoffmann type. It differed from this in that sensation was entirely abolished to the level of the second rib, and also in the age at which the symptoms began. The condition developed *in utero*, and the child died when it was nine weeks old, so that the disease in this family began earlier and was very much more rapid than in other recorded cases. Pathologically, the case was like the recorded cases in that the cells of the anterior horns were extremely atrophied, but it differed from them in that the posterior columns were degenerated. Other children in the family were affected.

The paper of Finkelnburg [26] makes the resemblance of certain cases of amyotonia, such as those of Spiller, Lereboullet and Baudouin, and Councilman and Dunn, to muscular dystrophy striking. As Finkelnburg says, in spite of the immense amount of work done regarding muscular dystrophy, anatomical investigation of cases in the first months of life did not exist previous to his study. A microscopical examination of dystrophic tissue in early life, he thinks, is of great value in the disputed question of neuropathic or myelopathic nature of the disorder. His case was as follows: A boy, aged 21 months, developed normally until the eleventh month, according to the mother's statement. He could sit alone at the sixth month, and attempted first to stand at the ninth month. At the eleventh month the mother noticed the child had lack of muscular tonicity. In sitting he sank forward, so that he must be held by the shoulder-girdle. One can hardly suppose from these statements that the child was normal until he was 11 months old; such weakness would be gradual in development. The disease must have been congenital or acquired in the first months. The calf and gluteal muscles were especially well developed, although very soft, in contrast with the atrophied upper arm and shoulder muscles. The limbs were weak, especially in the proximal parts. The tendon reflexes of the lower limbs were not obtained. The child lived only three days under medical observation. Two other children in the family had muscular dystrophy at the eighteenth and twenty-fourth month respectively. Carey Coombs's case of amyotonia is interesting in this connexion inasmuch as the upper limbs were more affected than the lower.

In this case of Finkelnburg there is much to suggest amyotonia from a clinical point of view. The findings suggested muscular dystrophy, but there were, on microscopical examination, fields composed entirely of very small muscle-fibres without increase of connective tissue between them. Finkelnburg thought these small fibres could not be considered as normal fibres in a state of development, as they were much smaller than they should be in a child of the age of his patient.

Councilman and Dunn, however, have shown that muscle-fibres vary greatly in size in children of the same age. Finkelnburg concluded that the small fibres were not atrophied fibres, but fibres arrested in their development, and their tendency to dystrophic change was shown in the presence of hypertrophied fibres among the small fibres. No changes in the nerve-cells were found.



FIGS. 1, 2, and 3.—Congenital weakness and hypotonia with "winged scapulæ" and "loose shoulders."

Dr. Langley Porter, of San Francisco, California, has given me the notes of a very interesting family in which the diagnosis between amyotonia and muscular dystrophy is not altogether easy. The disease is a familial one.

The parents of the children are healthy, and no similar disease is known in the ancestry. A maternal grandfather had syphilis. The family consisted of five children. The first child, a girl, is now aged 14, well developed and normal. The second child, a boy, had a condition which may have been amyotonia or muscular dystrophy with rickets. He had recurrent attacks of bronchitis, and died in one of these. He was never able to sit erect, to hold the head erect, or to raise the upper limbs above the shoulders. No electrical reactions were taken. The third child, a girl, now aged 9, is in perfect health. The fourth child, a boy, died a few days after birth, and possibly had an atonic condition. The fifth child, a boy, is now aged 3 and a few months (April, 1911), figs. 1, 2 and 3. He did not use his hands as infants do; he fixed his eyes on objects, but did not reach out to grasp. He did not hold up his head until he was more than a year old. He was not able to sit erect until he was 20 months old. He has never shown any inclination to stand. When 14 months old, he did not use his hands well; now he uses them very well. He has no power in the shoulder-girdle, and the muscles of this part, the erectors of the spine, and all the mucles of the hips and thighs are small, so that all these groups appear smoother and flatter than normal, though not distinctly atrophied. He has distinctly "winged scapulæ" and "loose shoulders." He can flex the feet to a right angle and move the toes freely. He sits on the buttocks and twists himself from side to side in moving over the floor. Hypotonicity is pronounced, so that the front of the thighs can be placed against the trunk. The boy is very intelligent.

In a letter received from Dr. Porter nineteen months after the above notes were taken, it is stated that the boy has shown steady improvement in power of all the muscles other than those of the shoulder and hip-girdle groups, and of the neck. He still has difficulty in supporting his head, especially when he loses its balance, at which time it drops on to the chest, but never rolls sidewise nor backward. He can hold himself erect if supported by leaning against some object, but has no power to stand independently. He is subject to frequent attacks of bronchitis, and it is evident that the respiratory musculature is weakened.

The weakness in this case was congenital; this fact and the progressive improvement convinced Dr. Porter that the case is one of amyotonia, notwithstanding the familial character of the disease. On the other hand, the marked weakness of the muscles of the shoulder-girdle, the winged scapulæ and loose shoulders are suggestive of muscular dystrophy. Remarkable for dystrophy is the congenital commencement and the hypotonicity, so great that the front of the thighs can be placed against the trunk. Whatever view may be entertained regarding this interesting family, it shows clearly that a diagnosis between amyotonia and dystrophy may be difficult.

It thus appears that it is impossible to maintain that in amyotonia there is:—

(1) Absence of any familial tendency in the incidence of the disease.

(2) Absence of any familial or ætiological relation with myopathy.

The question now to be answered is: Are there any cases of myopathy in which the disease was present at birth?

If Dr. Porter's case be one of myopathy, as I am inclined to believe, the answer to this question is in the affirmative. Finkelnburg's case was one of very early myopathy, and possibly earlier than is recorded. Kaufman Schlivek [49] reports a case of congenital myopathy. The anterior part of the arms was flat, soft, and flabby. The posterior part was better developed, but also soft and flabby. The shoulders and forearms were well developed. Active motion was limited at the shoulder; pronation and supination of the forearms was good. Motion in the wrists, hands, and fingers was normal. The muscles of the thighs and legs were soft and flabby. There was lordosis when the child stood, but not when she was sitting. Knee-jerks were present. Electrical reactions showed only quantitative change. The author considered and excluded amyotonia.

Russell Howard [37] reported a case of congenital defect of the muscular system (dystrophia muscularis congenita), and its association with congenital talipes equino-varus. The child was one of twins, and lived seven days. The report is a pathological one. The spinal cord was not examined microscopically, but peripheral nerves were examined, and were normal. The case was regarded as one of primary degeneration of the muscles occurring before birth. The writer believes congenital talipes may be a limited congenital muscular dystrophy.

It is doubtful whether either amyotonia or myopathy ever begins acutely or reaches a maximum within a few days. Batten thinks a development of this kind is rare, and the cases in which it has been recorded can hardly be taken as proof of its occurrence. He gives no references to any cases of this character, and states that it is admitted that an acute illness may bring into prominence symptoms which have not been observed previously; but it has not been proved that the true hypotonic condition of the muscles seen in typical cases of amyotonia is acutely produced.

Collier and Wilson, [14] however, assert that in three cases the condition came on rapidly, and reached its maximum within a few days of the onset of an acute illness in a previously healthy child. In one of their cases an attack of acute bronchitis was followed by complete paralysis within a week in a child aged 12 months, who had been until this time apparently healthy. In Leclerc's case a healthy child, aged seven weeks, was seized with broncho-pneumonia, and became paralysed generally within a few days, and though slow improvement had gone on all the time, four years later the case was typically one of amyotonia. In Comby's case the amyotonia came on rapidly, following an attack of diarrhœa at four weeks, and was almost universal in its extent, and severe in degree. Collier and Wilson say the histories of these three cases hardly admit of the explanation, that we are here dealing with the effect of an acute illness in bringing into evidence an amyotonic condition already present in minor degree, but it suggests strongly that an acute toxic process was the cause of the amyotonia.

Leclerc [41] was doubtful of the correctness of the parents' statement concerning the rapidity of the onset. He says the child appeared normal at first, but at the age of seven weeks had broncho-pneumonia and spasm of the glottis, and the muscular condition then developed. He asserts, however, that it was a typical case of amyotonia, that it was almost certainly congenital, even through the parents believed the spasm of the glottis indicated the onset of the amyotonia. He thought the parents overlooked the early condition. At all events, the increase of the symptoms seems to have been sudden and severe.

Comby [16] says of his case that the child was born at term and well formed. It was noted soon that the superior or inferior limbs, sometimes all the limbs, even the head, could not be moved freely.

It is doubtful whether these two cases can be accepted as demonstrating an acute onset of amyotonia, and the possibility of such an onset depends alone on the report of the case of Collier and Wilson.

We can hardly accept the case of Armand-Delille and Albert Weil [1] as one of myopathy, even though it was so regarded by them and by Raymond. The rapidity of development and the recovery make the diagnosis very questionable.

The acutely developing cases of amyotonia may be confused with anterior poliomyelitis and diphtherial palsy. Collier and Wilson say so far the acute cases have followed only acute bronchial disease. As a rule poliomyelitis may be distinguished, according to these writers, by the strict symmetry of amyotonia upon the two sides, the absence of local muscular atrophy, the absence of complete paralysis, and the absence of the rapid narrowing down of the extent of the regions affected which follows an attack of infantile paralysis. Granting that these points will enable poliomyelitis to be distinguished from amyotonia, there are cases of poliomyelitis in which there is symmetrical or nearly symmetrical and general implication of the lower limbs or of all four limbs, the wasting is general in the affected parts, the paralysis is complete in the affected muscles, and partial recovery is slight or absent. It is true that such cases are the exception, and that usually poliomyelitis can readily be distinguished from amyotonia congenita.

Diphtherial palsy may cause even more confusion should acute cases of amyotonia become more common. Collier and Wilson state that diphtherial palsy is very rare during the first year of life, when amyotonia usually develops. The absence of deep reflexes and the flaccidity may be in both, but the presence of nasal regurgitation and of cardiac debility, and the history and cause of the diphtherial palsy should render impossible the making of any mistake between the two diseases in the opinion of Collier and Wilson.

We can hardly dispute, however, that diphtherial palsy may occur with so little pharyngeal involvement that it may be overlooked, and nasal regurgitation and cardiac debility may not be conspicuous in all cases of diphtherial palsy. Again, it is to be remembered that the attempt is to be made to distinguish diphtherial palsy, rare in children of the first year, from an even rarer affection, acutely developing amyotonia.

Diphtherial palsy becomes especially important in connexion with Bernhardt's [9] views regarding amyotonia. He believes a multiple neuritis or an arrest in the development of the nerves is more likely to be the cause of amyotonia than an arrest in the development of the muscles. The improvement which occurs in this disease can be explained better on nerve disturbance than on muscle disturbance. He acknowledges that disease of, or deficient development of, the nerves in amyotonia has not been determined. He has shown that multiple neuritis may occur in the first years of life, and the cases he reports bear a striking resemblance to amyotonia. Slow contraction of the affected muscles in amyotonia by direct galvanic current, he says, was observed once by Oppenheim and in one of his own cases. We must therefore acknowledge that multiple neuritis developing in the first year of life, at least as early as the fourth month, may cause a clinical picture apparently indistinguishable from amyotonia, and the neuritis is not necessarily from diphtheria. In one of Bernhardt's two cases recovery strengthened the diagnosis of neuritis, as recovery would be less likely to occur in a disturbance of the muscles than in one of the nerves.

A case which I had the opportunity to study in Dr. Griffith's wards presented the appearance of an acutely developing muscular dystrophy, and is similar to the case reported by Armand-Delille and Weil. The complete recovery after some weeks suggests multiple neuritis, and yet pain was never a prominent symptom, and objective disturbance of sensation never occurred.

J. G. (figs. 4 and 5) was admitted to the University Hospital on March 19, 1912. His age was seven years. Two brothers and one sister were living and well, and no children had died in the family. The patient had not used alcohol, and alcoholic neuritis is very rare in children. He had had no symptoms of diphtheria. His teacher had noticed that he had been very dull mentally for two months. He had masturbated. He went to school until about six weeks before he came to the hospital, and he was then noticed to be walking slowly. He was said to have fallen on the steps three weeks before admission, and following this had pain in both lower limbs and the right upper limb. It was difficult to judge concerning the character of this pain. It did not appear to have been severe, and he had none when he came under the observation of physicians. It may have been from bruising of the muscles in the fall, or it may have been from use of weak muscles, and it is even possible that the statement concerning pain was incorrect. The weakness antedated the fall.

I found the pupils equal, and the irides reacting normally to light and in accommodation. The limbs were rather massive, suggesting the possibility of pseudo-hypertrophy. When he was placed at full length on the floor and told to raise himself, he turned over on the knees and palms of the hands, placed the right hand on the right thigh, but was unable to take the left hand from the floor and to raise the body above this position. When he was allowed to sit on the floor the muscles of the back at times were too weak to support him, and he fell backward and struck the head in the occipital region with great force. When he was lying in bed he could lift the lower limbs with difficulty, and could move them at all parts, but could not move them against slight resistance. The upper limbs were as weak as the lower, notwithstanding the apparently good muscular development. A brother of the patient, a little older, examined by me, was normal physically and mentally.

Extreme hypotonia certainly is not a common condition in myopathy, but Batten believes that marked flaccidity is present in many cases. I have never seen myopathy with the extreme hypotonicity of amyotonia, such as was present in my case of amyotonia, when the thighs

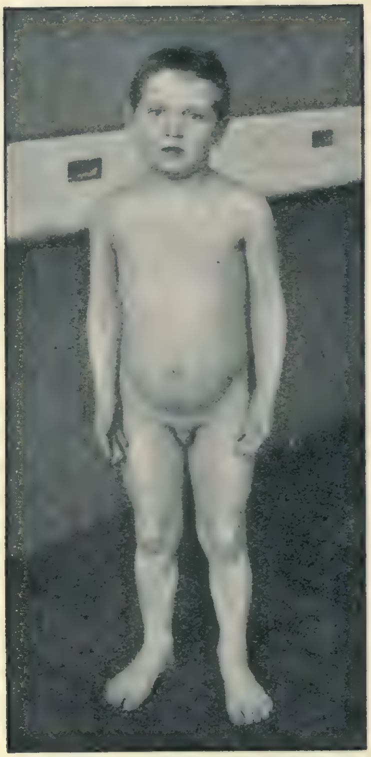


FIG. 4.—J. G. Excellent muscular development, but great weakness of rapid onset with recovery.

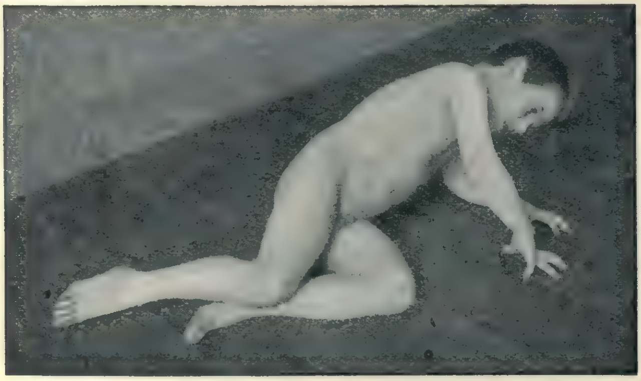


FIG. 5.—J. G. Showing attempt to climb on the lower limbs, as in pseudo-hypertrophic muscular paralysis. The photograph shows almost the full power of the boy. He was able to turn over a little farther than is represented, and to place the right hand on the right thigh.

could be placed close against the trunk and the heels behind the occiput; nor so intense as described by Collier and Wilson when they say that in several cases, including one of their own, the position which the foot assumed at rest was with the whole length of the dorsum of the foot lying along the front of the tibia. They state that the wrist and fingers can often be over-extended so as to come into complete contact with the back of the forearm.

Localized wasting is not always present in myopathy, and the atrophy may be general; still, localized wasting is a common feature of the early stage of myopathy, and its occurrence in amyotonia has not been observed. General wasting may occur in amyotonia, as in Thorspecken's case [57]. This case was typical in the absence of family disease, in the hypotonia, and of electrical reaction and in improvement. Atypical were general localization and symmetry of the paresis, the condition of the reflexes, muscular atrophy, the great atrophy of bone, and impairment of sensation, but this last-mentioned symptom seems doubtful. Cassirer says muscular atrophy does not belong to amyotonia, but Thorspecken finds smallness of muscles mentioned by many writers, and he thinks it is a fairly regular finding, and is masked by overgrowth of fatty tissue. The bone atrophy was found by the X-rays, and this method of examination has not been employed in other cases.

The untrustworthiness of progressive improvement in amyotonia as distinguishing it from myopathy is emphasized by Batten. As he says, in not a single case of amyotonia has recovery occurred; as the child develops, a certain gain of power has taken place, so that he may learn to stand or walk in a fashion. In Batten's case, D. S., improvement was said to be marked, but the boy could not stand alone, although he could do so with a little support. In many cases of amyotonia the condition has been progressive deterioration, not amelioration. In the case of amyotonia reported by Griffith and Spiller improvement for a time was marked, and this is remarkable in consideration of the pronounced and progressive changes of the nervous system, but, as in many other cases, the improvement was not permanent.

It is not because the period of observation in cases of amyotonia has been short that improvement is not great. In the case reported by Carey Coombs [17] the improvement after ten years was slight. The child was ten years old, and the condition had been present since birth. The upper limbs were more affected than the lower, and the hands were more affected than the rest of the upper limbs, so that there was localization in this case. In the study of any new disease experience teaches that certain statements may need modification, and that features considered sharply defined are of less importance than they at first seemed. Thus Collier and Wilson, writing in BRAIN (vol. xxxi, 1908), stated regarding amyotonia: "In no case has the least tendency to exacerbation of the disease been observed." In the next volume of BRAIN (vol. xxxii, 1909), these same authors, in reporting two cases, remark: "This is one of the only two cases that have come under our observation in which progressive deterioration has occurred." From the pathological point of view improvement appeared unlikely; they could detect no evidence of regeneration or of reparatory changes in the muscles.

On the other hand, myopathy may be arrested for a long period of years, or there may be improvement, usually temporary, and possibly obtained by enabling the patient to use the less affected muscles to better advantage, or by strengthening these muscles, or in rare instances recovery may ensue. Erb said he had seen only one case of recovery in myopathy. Marina's case he regards as not fully established as regards diagnosis. Erb's case [24] was a typical one of the juvenile type, and the patient was "practically cured" (as he expresses it) after six years, although some trifling defects remained.

Jendrassik [38] accepts Marina's case as one of myopathy, but he does not accept the case of Armand-Delille and Weil. He states that in Marina's case and also in Erb's the weakness was not great. He reports two cases of dystrophy with almost complete recovery. He says in all the cases the improvement occurred at the period of greatest physical development, i.e., at puberty; in Erb's, Marina's, and his own two cases the improvement occurred when usually rapid physical development takes place.

Gordon Holmes [36] reported a case of arrested myopathy. A man, aged 60, was found to have extreme weakness of the orbicularis palpebrarum of each side, slight weakness of the orbicularis oris, and the cheeks were remarkably flat and thin. Since he was 10 years of age he had not been able to whistle properly. The muscles which depress the arms were weak, and there was considerable wasting of the lower part of each pectoralis major, and atrophy of the upper portions of the trapezii. The man was unaware of the muscular abnormalities. His daughter and nephews had typical myopathy. Holmes regarded the case as one of myopathy which appeared early, but ceased to develop at a certain stage of its evolution.

Batten stated that in many cases of dystrophy it was admitted that there was evidence that the patients had had weakness of the face at birth, and when seen between the ages of 30 and 40 they still had the weakness of the face muscles, but no evidence of any progress of the disease.

Stanley Barnes has seen two cases of the Landouzy-Dejerine type; the patients were over 50, and in each there was no reasonable doubt that the pectoralis major had been absent for twenty-five years, and that the myopathy had not progressed for many years.

The only case of what seemed to be myopathy in which so great improvement occurred that it amounted to recovery which has come under my observation is the following: A man, aged 24, consulted me on October 27, 1904. None of his brothers or sisters had a similar disorder, although he had five brothers older than himself. He may have had typhoid in 1902, but it is not certain. When I saw him he had been complaining of weakness in the right lower limb about a year and a half. The muscles of the anterior part of the left thigh had seemed weak to him, but the weakness was not pronounced. During the three weeks before consulting me he had had moderate pain in the anterior muscles of the right thigh, probably from over-use of weak muscles, as he never had it before this time or later. He had lost about 25 lb. within the previous two years. He had much difficulty in walking, and could walk only a square or two.

On examination, the thighs were equally well developed, and measured about 16 in. in circumference in the middle. The right calf was large, and much larger than the left, which appeared atrophied. The circumference of the middle of the right leg was 13½ in.; of the left leg, 12½ in. The left calf was soft and flabby; the right calf was very firm. He could not stand on the right foot alone as he could on the left foot.

Flexion of the right thigh on the abdomen was somewhat impaired, and flexion of the right leg on the thigh and pushing with the right foot against the hand were a little impaired. The patellar reflex was present on each side, but was not very prompt. The Achillis reflex was hardly perceptible on the left side, and was lost on the right side. Sensation was not affected in any part of the body. The trunk, upper limbs, and face were not affected. Faradic contractility was prompt in the muscles of the left calf, but was lost in those of the right calf. Galvanic reactions were not taken. He was given massage.

This patient was last seen by me on June 13, 1912. The extraordinary firmness of the right calf had almost disappeared, and only a small mass of unusually firm tissue, about the size of an English walnut, remained. All weakness had disappeared, and he could walk fifteen miles daily with ease. The Achillis reflex was still lost on the right side. The patellar reflexes were nearly normal, possibly a little diminished. The peroneal supply on each side was unaffected.

The interesting features of this case were: weakness of the right lower limb, chiefly in the thigh, developing gradually during a period of one and a half years; possibly some weakness of the left thigh; apparently pronounced pseudo-hypertrophy of the right calf; atrophy of the left calf; diminished patellar reflexes; loss of Achillis reflex on the right side, and almost loss on the left side; absence of all objective and subjective sensory disturbances, except moderate pain in the right thigh in the weakened muscles of only three weeks' duration and probably from over-use; recovery during a period of almost eight years under massage and systematic exercise. The diagnosis of myopathy in this case would hardly have been disputed if recovery had not occurred. It is possible that typhoid fever may have been the cause of the weakness, and yet there was only an uncertain history of a disease that might have been typhoid; this had occurred two years before I saw him, and about six months before the onset of the weakness. The affection of both lower limbs made the diagnosis of thrombosis improbable. Hard patches in muscles are not uncommon in myopathy.

Typhoid fever may be a cause of myopathy, as Guillain has shown. He remarks that a few cases in literature have demonstrated the existence of true muscular hypertrophy as a result of typhoid. He gives the published cases to 1907 (Friedländer, Lesage, Cerné, Babinski). This enlargement of muscles appears to be a true muscular hypertrophy, with increased motor power, with diminution of faradic and galvanic contraction, without reaction of degeneration, and with exaggeration of tendon reflexes in certain cases, but not in all, and with limitation to one limb or part of one limb. The cause appears to be vascular lesions, especially phlebitis, and the condition an angio-myopathy.

Guillain distinguishes this hypertrophy from the functional or physiological muscular hypertrophy, the muscular hypertrophy of congenital myotonia, the muscular hypertrophy often associated with athetosis, the prodromic muscular hypertrophy of progressive atrophic myopathy, and the muscular hypertrophy of the hyperplastic muscular dystrophy of Talma.

In addition to this form of myopathy Guillain describes another form following typhoid fever, of progressive development and implicating many muscles, and he refers only to three cases: one of Friedländer, one of Josserand, and one of his own. The diagnosis of myopathy was confirmed by necropsy in Friedländer's case.

Guillain [32] believes he has observed many cases which demonstrate that myopathy may be acquired, and possibly from infection or intoxication. This is interesting in view of the cases of amyotonia developing acutely after pulmonary disease.

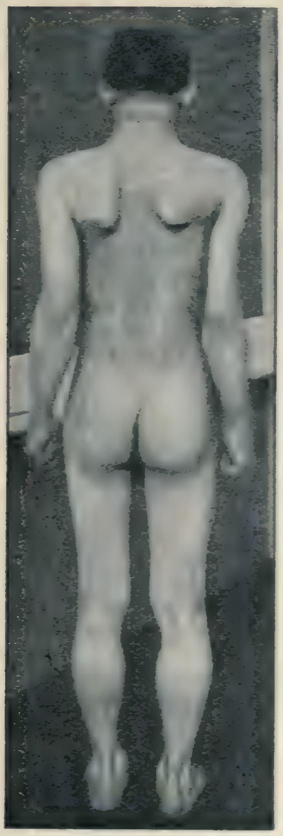


Fig. 6.—Myopathy with improvement. Atrophy of shoulder-girdle and upper part of upper limbs, "winged scapulæ."

I have known distinct improvement to occur in an advanced case of myopathy. E. S. (fig. 6) came to the dispensary of the University Hospital, on July 10, 1911. He was aged 19. A year previously he fell from a scaffold, a distance of sixty feet, and possibly struck on his shoulders. He was able to resume his work the same day, but the following day his arms were stiff and painful, and remained so for two days. From that time he could not lift anything heavy, and could do only light work. He noticed about four months later that the muscles of both shoulders were wasting, and that he had difficulty in using them. Since that time until the present the muscles have wasted, and he can raise the upper limbs to the level of the shoulders only. The deltoid, biceps, triceps, and pectoral muscles are much atrophied. No fibrillary tremors are observed. There are no sensory disturbances for touch and pain. There is no spasticity. The patellar reflexes are present, but not increased. There is no family history of similar trouble. A positive Wassermann reaction was obtained by Dr. Corson-White. Two brothers who were examined were normal.

I saw this man again in March, 1912. He was then under the care of Dr. John K. Mitchell. The atrophy conformed to Erb's juvenile type, and apparently had been started by the trauma, which is an interesting fact. No symptoms in the lower limbs suggested a cervical spinal lesion. Dr. Mitchell had ordered massage, and decided improvement had been obtained. There was distinct increase of power in the arms, hands, and legs. The man was able to comb his hair with his right hand, and this he had not been able to do before the treatment. He had found that the circumference of the upper limbs just above the elbows was 1 in. greater than it had been. He could raise the hands, when clenched, above the head. There probably has been no permanent arrest in the muscular atrophy, but important is the decided improvement in a case of myopathy, showing that improvement is not a feature of great value in the differential diagnosis between myopathy and amyotonia.

The failure of return of deep reflexes is common to both myopathy and amyotonia, and although return has been observed in the latter and not in the former, it is questionable whether great importance can be placed on this fact.

Failure of extension of the disorder from muscle to muscle in amyotonia at the present time appears as an important diagnostic sign, and yet who can tell how soon some one may report a case of amyotonia, with gradual spreading of the condition.

On the one side we have Batten urging the classification of amyotonia under myopathy, and on the other, Collier and Wilson and many others opposing this classification. Collier and Wilson remark that the similarity of the morbid anatomy of amyotonia to that of the myopathies is certain and does not admit of any discussion, but the clinical aspect of this disease is so widely different from that of any hitherto described form of myopathy as to warrant the retention of a separate name for what they call a distinct clinical entity. I have attempted to show in this paper that many of the so-called distinguishing features of amyotonia belong also to myopathy, but after all we must acknowledge that amyotonia has distinguishing features, observed in by far the greater number of cases, and that the cases with exceptions do little more than show that there may be a transition from amyotonia to myopathy, without proving that the two diseases are identical, any more than transitional cases between myelopathy and myopathy prove identity of disease.

There are a few cases of amyotonia which are typical clinically, and in which the lesions were confined to the muscular system; in order of publication these are the cases of Spiller, Lereboullet and Baudouin, and Councilman and Dunn. Possibly the case of Finkelnburg may be included. The pathology in these cases may be arrest in the development of the muscles with or without added muscular atrophy. Thorspecken [57] believes that not a single reported case of amyotonia can be regarded as one of arrested development, except possibly that of Archangelsky and Abrikosoff. In this case general and pronounced muscular atrophy of bone and contractures showed much resemblance to myopathy. Increase of connective tissue nuclei and of interstitial fat tissue are more in favour of disease changes in fully developed elements, but in my opinion do not exclude the possibility of arrest in development, at least as a part of the process. One may readily believe that tissue arrested in development would be more liable to degeneration.

In my case [53] increase of connective tissue nuclei and of interstitial fat tissue was relatively slight, and Councilman and Dunn in the report of their case show how difficult it is to judge of the intensity and importance of such findings. My case was a typical one of amyotonia, and although objection has been made to it by a few writers, it is very evident that some of these writers have never read my papers. It is impossible, for example, to reply to a statement that a case "nicht über allen Zweifel erhoben ist," or "nicht ganz typisch ist," but when Gött and Schmidt state that the child I described was idiotic and had strabismus and amaurosis, I can only reply that they know more about the patient than I do. There is no remark in my papers that suggests idiocy in this child, and I have shown by a statement from Professor de Schweinitz of the Ophthalmological Department of the University of Pennsylvania that the ordinary concomitant squint present in the case was not the strabismus from ocular palsy, and that it is very doubtful whether the child was blind. This case was carefully studied clinically and by microscopical specimens, and microscopical sections were made from various levels of the spinal cord, from muscles from the sole of the foot, calf, back of the trunk, and arm, and from peripheral nerves.

The case of Lereboullet and Baudouin cannot be rejected merely because convulsions occurred, and that during these convulsions the muscular atony disappeared. The patellar and olecranon reflexes were diminished, but not lost. The exaggeration of reflexes later in the course of the disease is not sufficient to exclude amyotonia, and it was probably the result of the convulsions. A child with amyotonia is not exempt from convulsions.

The findings in the case reported by Councilman and Dunn [19] were purely muscular. The amount of connective tissue was increased. Some fibres appeared like normal adult muscle-fibres, others were very small, and under low power appeared as accumulations of cells arranged in fasciculi. The nuclei in the small muscle masses seemed to be extremely numerous. The apparently great number of nuclei was due to the number of the fibres, and when a single fibre was followed along the nuclei were not more closely set than they were in the large fibres. Occasionally fibres were seen which were hollow, with fibrillæ at the periphery, presenting much the appearance of embryonic fibres. There was no general fatty degeneration of the muscles, but in certain muscle-fibres there was a large amount of fat. The connective tissue in the muscles was relatively increased, but it was uncertain whether there was an actual increase in this tissue. The spinal cord was practically normal.

There are other cases of amyotonia with pronounced muscular changes and comparatively slight alteration of the nervous system, such as those of Baudouin, and Archangelsky and Abrikosoff. It is well to remember that while myopathy usually is without marked alteration of the nerve-cells of the anterior horns, even when the disease has existed so long as in the case reported by Duchenne [22] in 1872, by Landouzy and Dejerine [40] in 1886, and by myself [54] with the pathological findings in 1900, still there are transitional forms in which moderate changes of the anterior horn-cells are detected.

The subject of degenerative changes occurring in the lower motor neurones in muscular dystrophy has been discussed by Gordon Holmes. [35]. He reports a case of this disease in which there was considerable diminution in number and size of the nerve-cells of the anterior horns and atrophy and diminution of fibres in the ventral roots. He refers to a number of similar cases in literature. He thinks that in his case and in the majority of the others there can be no doubt that the condition was acquired, and was not a developmental defect. The nature of the cell-changes, the slight secondary gliosis of the ventral horns, the definite evidence of loss of fibres in the ventral roots and peripheral nerves, and the increase of connective tissue in the nerves, make this certain. He rejects the theory of nervous origin for the muscular atrophy in these cases, because the histological changes in the ventral horns in his case appeared to be distinct from the degenerative cell-changes he has found in any of the many spinal amyotrophies he has examined, and the muscular affection exceeded them both in degree and extent. He believes the neural changes are secondary to the primary muscular disease. He does not know of any evidence of neural degeneration secondary to disease entirely limited to muscle tissue, but he thinks it is probable that when muscle fibres undergo complete atrophy and disappear, the functional, and probably secondary thereto the nutritional, equilibrium of the neurones which terminate in these fibres must be disturbed. The terminal branches of the axis cylinders left naked by the disappearance of the muscle-fibres probably will be injured and possibly destroyed by the connective tissue which proliferates secondary to the muscle disease. He refers to Baudouin's case of amyotonia congenita, and shows how closely it resembles the case he reported. Baudouin himself spoke of the muscular changes, " telles qu'on peut les voir dans les myopathies." Holmes says that the morbid anatomy of Baudouin's case was identical with that of a considerable number of undoubted cases of muscular dystrophy. He states that the origin in his case and Baudouin's must have been similar, as Baudouin believed the abnormal condition of the muscles could not be regarded as merely an arrest of development, and must have been an acquired condition. Owing to its intensity the muscular alteration of Baudouin's case could scarcely have been secondary to the simple atrophy of the ventral horn-cells, and its nature, too, was distinct from that of the neuropathic atrophies. It must be concluded, Holmes thinks, that the disease of the muscles was an acquired regressive affection, dependent probably on an intrinsic developmental anomaly. From the pathological and clinical points of view Holmes thinks Baudouin's case differs from one of myopathy only in the commencement during intra-uterine life. The clinical manifestations so closely resembled those of myopathy that it seems unnecessary to place the case in a separate class. The most important distinction of amyotonia is that it is not progressive, and that amelioration may occur. Even if amyotonia were congenital myopathy, these distinctions would not be surprising in view of the difference of the nutritional conditions of the individual before and after birth, and the changes in the mutual relationship of the muscular and nervous systems in intra- and extra- uterine life.

It seems probable to me that an arrest with later atrophy may occur in the peripheral neurones as well as in the muscles in some cases of myopathy. It is common to find more than one defect in development. Oppenheim has emphasized this as regards myasthenia. Cataract with muscular atrophy is another example. The case of myopathy of long standing referred to above shows that great destruction of muscle does not lead necessarily to degeneration of the peripheral neurones.

Archangelsky and Abrikosoff regarded the changes in the cord in their case partly as arrest in development of the grey substance, and partly as a retrograde change. Most muscle-fibres were thin, but their structure was preserved; there was no nuclear proliferation of the muscle-fibres nor overgrowth in the connective tissue, therefore they regarded the muscular condition as arrested development. These findings in the muscles resemble those in the cases of Spiller, Finkelnburg, Lereboullet and Baudouin, and Councilman and Dunn, but Archangelsky and Abrikosoff found diminution in size of the anterior horn-cells, and this finding is like that in the case of Baudouin.

It seems, therefore, that the symptom-complex of amyotonia may depend on purely muscular lesions (Spiller, Lereboullet and Baudouin, Councilman and Dunn), or these muscular lesions may be associated with alteration of the cells of the anterior horns, which in some instances seems to be slight (Lereboullet and Baudouin, Archangelsky and Abrikosoff), in others intense, and when this cellular alteration is intense the distinction from the Werdnig-Hoffman type of atrophy may be difficult.

THE RELATION OF AMYOTONIA TO THE WERDNIG-HOFFMANN TYPE OF ATROPHY.

Rothmann [47] has considered this aspect of the subject. Heredity, progression of the disease, reaction of degeneration, and commencement in the proximal parts of the limbs are the most distinguishing features of the Werdnig-Hoffman type. This form has occurred without familial character, as in the cases of Thomson and Bruce, Bruns, Wimmer, Armand-Delille and Boudet. In amyotonia Rothmann believes the spinal process has reached its height at birth, and improvement depends on the condition of the persisting anterior horn-cells. The Werdnig-Hoffmann atrophy, beginning in the second half of the first year or later, has a more gradual destruction of the nerve-cells, and yet Rothmann acknowledges that amyotonia beginning in the first months of life may show much improvement. The improvement in one case, the progression to death in the other are not sufficient to distinguish the two processes. The progression to death may be greatly delayed, as shown in three of Bruns's cases. Rothmann does not regard the reaction of degeneration as an important distinction, and this subject will be considered a little further on in this paper. As for the pathological findings Rothmann thinks they show gradations.

Rothmann's conclusions are :—

Amyotonia in the majority of cases anatomically is a disorder of the anterior horn-cells, and these cells may completely disappear.

All gradations exist between amyotonia and the Werdnig-Hoffmann type, so that sharp distinctions cannot be made between these diseases.

Transitional forms occur between the spinal muscular atrophy of fœtal origin (amyotonia) with affection of the lower cranial nerve-nuclei, and the nuclear loss of Möbius ("Kernschwund") with implication of the spinal cord.

Cassirer [12] regards as important distinctions that amyotonia is not progressive, and the Werdnig-Hoffmann atrophy first is seen in children in the second half of the first year of life. It is questionable whether these can be regarded as important. It is difficult to say exactly when a disease begins, especially when the date of its commencement is often determined by a more or less ignorant parent, and Wimmer's case [58], which he regarded as belonging to the Werdnig-Hoffmann type, was congenital. Cassirer is not willing to class amyotonia under spinal affections, and in his opinion it is closer to myopathy.

The cases of amyotonia with intense alteration of the cells of the anterior horns seem to indicate that the disease may be without amelioration, and even may be progressive. Such cellular changes are usually progressive in spite of all we do to check them.

Batten [4] acknowledges the difficulty in distinguishing between amyotonia congenita and the Werdnig-Hoffmann paralysis. His points of distinction are that in amyotonia a child as a rule can perform all movements in a feeble manner, and there is not the marked degree of flaccid paralysis seen in the case of spinal atrophy. Although hypotonia is present in cases of spinal atrophy, it is not so marked as in cases of amyotonia. The intercostal muscles are commonly paralysed in the spinal atrophy of infants, whereas in amyotonia they are less affected. In brief, in spinal atrophy the paralysis is more marked and the hypotonia less marked than in cases of amyotonia.

It is indeed a question whether further experience will justify these distinctions. They depend upon degree rather than upon quality of characteristics, and do not apply to some of the cases of amyotonia described with intense pathological change of the anterior horn-cells. In a mild case, one in which the spinal changes have not advanced far, and especially one in which the lesions are purely or largely muscular, all movements may be possible.

The flaccidity has been very great in a number of cases of amyotonia, and loss of power has amounted to complete paralysis (Archangelsky and Abrikosoff). In some of the cases of the Werdnig-Hoffmann type the flaccidity has been pronounced, and was so in a case reported recently by Batten and Holmes [8]. These authors had made a diagnosis of the Werdnig-Hoffmann type, but some who had seen the patient had regarded the disease as amyotonia. It is difficult to understand how the intercostal muscles could be little affected when the changes in the anterior horn-cells of some of the cases of amyotonia are so great. One is reminded by these distinctions of the attempt to separate chronic poliomyelitis from progressive spinal muscular atrophy.

The intercostal muscles have been reported as affected in a number of cases of amyotonia. Lugenbühl [42] remarked that the muscles of the thorax and neck were badly developed, and were not active. Pollak [46] said of his case that the thorax and back muscles were paralysed.

In the case reported by Griffith and Spiller [31] there was no history of acute disease. The mother said the boy had never been able to move his legs effectually or to handle objects. He took slight notice of anything, and had never been able to sit up. The legs were motionless and flaccid, the feet in position of foot-drop. The muscles of the legs were soft, but without actual atrophy. The toes and legs could be moved freely by the child when he was stuck with a pin. Passive movement at the knee-joint was unusually free, but there was contracture at the ankle. The muscles of the arms were soft, but not atrophied. The child was able to move the arms very feebly and slowly, and with little ability to handle objects. No tendon reflexes were obtainable. He was unable to sit unsupported, and when held in a sitting position extreme kyphosis developed, and the head dropped in any direction in which gravity acted upon it. When released the trunk would fall in a similar manner, sometimes forward, with the head against the ankles and the arms lying helplessly where they happened to be, and the child was unable to change this position.

Two and a half months after the first observation the intellectual power was found improved, the child took more notice of objects, the power in the upper limbs had increased, and he could now grasp a biscuit and eat it unaided. All movements of the upper limbs were still slow and feeble. There was slight increase of movement in the lower limbs. Under electrical or other stimulation there was decided increase of movement in the limbs, but no distinct muscular contraction was obtained by electricity, faradic or galvanic.

The findings in this case were very similar to those of Rothmann and those seen in the Werdnig-Hoffmann atrophy. I should like to refer to the fact that this child, as in the previous case reported by myself, took little notice of objects, as in no way indicative of poor sight. A child so weak has little interest in what is going on about him.

In the case reported by Gött and Schmidt [27] the clinical picture resembled amyotonia more than the Werdnig-Hoffmann type, except in certain features, viz., the absence of improvement, with a tendency to increase in the symptoms, the presence of reaction of degeneration (this is said to be always absent in amyotonia, but in Thorspecken's case slow reaction was obtained), and the presence of fibrillary tremors (these have never been seen in amyotonia, and are regarded as rare in the Werdnig-Hoffmann atrophy). Opposed to the Werdnig-Hoffmann type were the absence of heredity and familial tendency, and the appearance of symptoms immediately after birth, and not in the course of the second year of life. The findings were those of the Werdnig-Hoffmann type. Gött and Schmidt try to answer those who regard amyotonia and the Werdnig-Hoffmann atrophy as the same disease, but the very case they report is in support of a close relationship between the two. The cases of Wimmer, and Gött and Schmidt show that a congenital form of spinal muscular atrophy exists. The latter case is important, for it had the symptoms of amyotonia, including the hypotonia, and other signs showing clinically implication of the spinal cord. The muscles were much degenerated, the nerve-cells of the anterior horns much diminished in number and size, and the anterior lumbar roots were not so well medullated as the posterior.

Marburg [43] also declines to accept amyotonia as identical with the Werdnig-Hoffmann atrophy. He lays much stress on the absence of the fatty pigmentary degeneration of the nerve-cells in the Werdnig-

Hoffmann atrophy, which is so common in the Aran-Duchenne type, and has never been seen in very early childhood. Since his paper was written, Zatelli [60] has reported the finding of distinct fatty pigmentary degeneration of these nerve-cells, recognized by the Marchi stain, in a case of the Werdnig-Hoffmann atrophy. This seems to be the only case in which this change of nerve-cells has been found in children.

The distinctions Marburg makes are:—

Amyotonia.	*Werdnig-Hoffmann Atrophy.*
Congenital.	Acquired early.
Usually single case.	Usually familial.
General hypotonia.	Localized atrophy, beginning in pelvic girdle.
Atrophy concealed.	Atrophy easily recognizable.
Loss of tendon reflexes.	Tendon reflex activity in proportion to the atrophy.
Electrical irritability (?) or amyotonic reaction (faradic lost, galvanic normal).	Reaction of degeneration.
Improvement.	Progression with constant impairment.

These features, Marburg holds, are the most important, and show the differences between the two diseases. The congenital origin, it has been shown, is not always present in amyotonia, and Wimmer's case of the Werdnig-Hoffmann type and the case of uncertain classification of Gött and Schmidt were congenital. Familial cases are infrequent, but probably occur in amyotonia, and many cases of the Werdnig-Hoffmann type have not been familial. Hypotonia may be pronounced in either affection, although it is likely to be more intense in amyotonia. The atrophy in amyotonia may be distinct, and may implicate the bones, as in the case of Thorspecken. The condition of the tendon reflexes as a point of distinction is unreliable, especially in such young children. Galvanic and faradic reactions were lost in the case of Griffith and Spiller. In Rothmann's case strong faradic irritation gave only slight contraction of the flexors of the forearm, galvanic gave slow contraction, but KClC was greater than the AnClC. This case was studied in Oppenheim's clinic while the child was living, and was accepted as one of amyotonia.

As regards electrical irritability, Cassirer states that the determination of the electrical reactions in amyotonia is difficult in so young children. Some investigators have found that the children endure strong currents easily, but this Cassirer and some others dispute. In no case has a very exact electrical examination been made; according to Cassirer, in general it may be said there is diminution of the irritability, but no qualitative change. A peculiar reaction has been described by Collier and Wilson, and this they call the amyotonic reaction; the muscles react much below normal to a strong faradic current, whereas to the galvanic current the formula and form of contractions are normal. Cassirer says his experience shows that this difference in reaction to the two currents is not present in all cases.

All the muscles and nerves reacted to the faradic and galvanic currents in Thorspecken's case, but to stronger currents and in narcosis. Without narcosis the reactions were uncertain in some of the muscles. Slow reaction to the galvanic current was obtained in two muscles. Oppenheim is said to have had a similar finding in one case of amyotonia.

Improvement is not confined to amyotonia, and in many cases of this disease has not been pronounced, as has already been stated.

It would seem, therefore, that the distinctions given by Marburg are not so positive as they appear at first sight. He regards amyotonia as fœtal acute poliomyelitis, but he acknowledges that the existence of fœtal poliomyelitis has not been positively demonstrated. I do not know of any other writer who has accepted this view. I have seen nothing in the two cases I have studied that would justify such an opinion, and it is disputed by Thorspecken, who distinguishes amyotonia by the great extent and symmetry of the muscular disorder, by the absence of complete loss of function in a single muscle, by the characteristic electrical reaction (of somewhat questionable value), by the absence of vasomotor and trophic disturbances, and by improvement after a period of years.

Batten has attempted to demonstrate that intra-uterine poliomyelitis may exist. The first case reported by him in his paper does not afford absolute proof of fœtal poliomyelitis, but makes it probable. Especially characteristic of poliomyelitis is the elongated patch with almost complete disappearance of medullated fibres in the right ventral horn of the sacral region, represented in fig. 4 of Batten's paper. A patch of this character in the ventral horn, consisting of neuroglia, and without medullated nerve-fibres or nerve-cells, is very suggestive of poliomyelitis. Marburg's view that amyotonia is fœtal poliomyelitis receives some support from Batten's case [5], but in none of the cases of amyotonia have findings exactly like these been described, in none were these characteristic patches of neuroglia tissue representing scar formation following an inflammatory exudate.

THE DISTAL TYPE OF MYOPATHY.

Cases of the distal type of myopathy have been reported by Oppenheim and Cassirer [45], Gowers [28], Dejerine and Thomas [21], Spiller [55], Batten [6], and Cottin and Naville [18]. Campbell's case [10] is somewhat doubtful. To this list I now add another case, although it is without necropsy.

The case of myopathy of the Aran-Duchenne type (the atrophy began in the hands), reported by Merklen and Schaeffer [44] was purely clinical. Reaction of degeneration was present in some muscles of the lower limbs.

In my paper in which a case of the distal type of myopathy was reported, I emphasized the importance of recognizing as belonging to the peroneal (Charcot-Marie-Tooth) type the long-continued and sharp limitation of the atrophy to the parts below the knees and elbows. Cases in which the thighs and upper arms are wasted may belong to this type but they are not questionable.

Batten thinks the disturbance of peripheral sensation, often found in the peroneal type, points strongly in favour of the peroneal type as distinguishing it from the distal type of myopathy, and implication of facial muscles is a point strongly in favour of the muscular origin of the disease.

Hoffmann, however, accepts involvement of the facial muscles as a part of the peroneal type, as Batten mentions, and in Oppenheim and Cassirer's case of myopathy implication of the cranial nerve-supply was slight. In myopathy the atrophy may begin in the distal parts of the limbs, but sooner or later extends beyond them, and the trunkal muscles are likely to be affected. Sensory disturbances are so often absent in the peroneal type that in many cases the condition of sensation affords no means of differential diagnosis.

Cottin and Naville believe that in peripheral myopathy, as regards the hand supply, the atrophy has not the same distribution in the beginning of the disease as in the Aran-Duchenne atrophy. In the peripheral myopathy the hands fall and the fingers are flexed, and atrophy appears later, whereas atrophy is always early in the Aran-Duchenne form, and does not effect the extensors of the thumb and fingers until late. Peripheral myopathy, like the juvenile form, affects especially the extensors. These authors believe that peripheral myopathy begins late (middle life), is not hereditary nor familial, and is more common in females. In my case it began about the age of 26 years, and was in a male. In

Gower's case it began at the age of 10 or 12 years, and was in a male. In myopathic families females are affected later than males, and less severely, or even escape. The frequency of the peripheral myopathy in women affords a contrast to the rarity in women of the Aran-Duchenne and Charcot-Marie-Tooth types of atrophy, in which the distribution and time of onset are much the same. These conclusions of Cottin and Naville are untrustworthy, because they employ three cases only, and ignore those of Gowers, Spiller, and Batten. Gowers's case is referred to as an atypical form. It would seem from the work of Cottin and Naville that in the peripheral type of myopathy the extensors of the hands and fingers and the extensors of the toes are chiefly affected. In Batten's case the patient was a boy aged 3 at the onset of the symptoms. Another case now reported by me is similar in that a young boy was affected.

The boy described by Batten was well until the age of 3, when he contracted measles. A few weeks later the mother noticed dragging of the left leg. Seven months later the right leg became affected, and power in the hands began to be lost at the age of 6. The wasting of the arms and legs progressed slowly. At the time of examination (18 years of age) there was much wasting of all the muscles below the knee and a considerable amount of wasting of the thighs. The foot was in a inverted position, and the boy walked on the outer side. There was marked wasting of all the muscles of the arms below the elbow, and but little power of movement in the hands and forearms. The upper arms and shoulders were well developed. The muscles of the face and trunk were normal. The deep reflexes of the leg were absent. There was no response to faradism or galvanism in any of the muscles below the knee or elbow. Sensation was normal. There was no marked vasomotor disturbance in the legs. Batten, some years previously, had shown this case as one of the peroneal type following measles. At the time of the presentation of this patient he reported the occurrence of the distal type of myopathy in several members of one family. These cases called forth considerable difference of opinion. Holmes remarked that the age of onset of peroneal palsy was higher than in Batten's cases.

A recent case which I have studied is as follows: T. B. (figs. 7 and 8), white, aged 9, came to the Nervous Dispensary of the Hospital of the University of Pennsylvania on June 27, 1911. The first child was born at the sixth month, and died when a few months old. The patient also was born at the sixth month. The labour was normal. He weighed five pounds at birth, was very small, and developed slowly. When he was 3 years old he gradually developed weakness and deformity of the ankles and legs, but there is some doubt whether the weakness began in the ankles or in the thighs; the much greater atrophy of the legs would suggest that the atrophy began in these parts. He was very healthy when 2 years old, and better developed than most children of the same age.

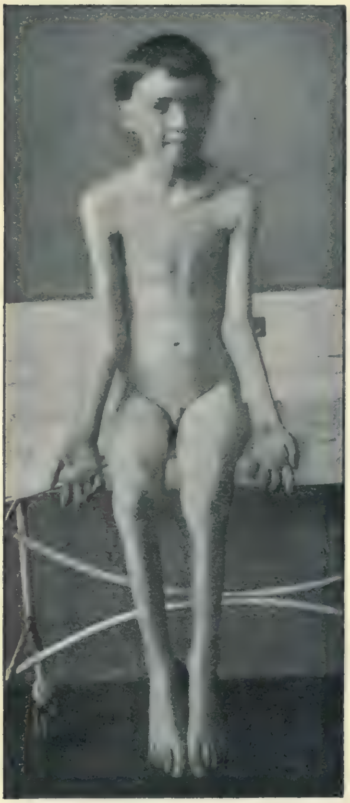


FIG. 7.—Distal type of myopathy. The photograph shows the bilateral foot-drop and greater wasting of the lower limbs below the knees and atrophy of hands and forearms.

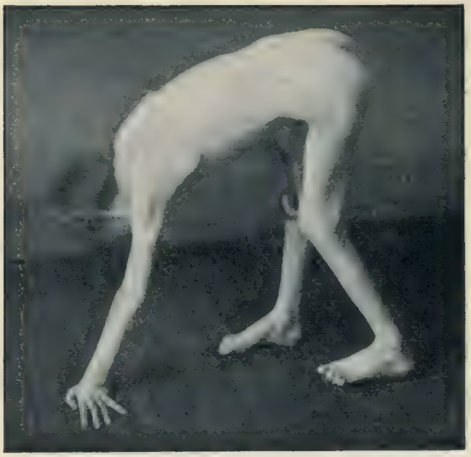


FIG. 8.—Distal type of myopathy. Photograph shows the extent to which the patient was able to rise from the floor. The method of rising is like that of pseudo-hypertrophic muscular paralysis.

In June, 1911, the head was not affected apparently. The movements of the hands were somewhat awkward and ataxic, probably from weakness, and the muscles of the right hand appeared as if they were beginning to atrophy. Both scapulæ were prominent and winged.

The muscles of the back and thorax were small, but there was no localized atrophy in these parts. The feet were placed far apart, and there was grave foot-drop on each side, and steppage gait. When rising from the floor he climbed upon his lower limbs. There was symmetrical atrophy of all the muscles below the knees. The calf muscles seemed stronger than the others. Sensation was normal for pin-prick, but the examination was not satisfactory in this respect. The patellar reflexes were absent and there was no ankle-clonus. The big toes did not move upward in testing for the Babinski reflex. The Wassermann test was negative, the Noguchi positive.

In March, 1912, the atrophy of the small muscles of the hands was pronounced, and much more so than in June, 1911. In raising himself he could not take both hands from the floor.

The boy was examined by me again on December 26, 1912. He was able to walk without support. The lower limbs were weak and the gait was distinctly steppage, because of the bilateral foot-drop. The lower limbs were greatly wasted in all parts, and he was weaker than at my last examination. The middle of each leg measured 16 cm. in circumference. He had very little movement in the ankles and none in the toes. The legs were much more wasted than the thighs. The hands were very much wasted, and equally so. The thenar eminence had disappeared, and the hand was of the simian type, with over-extension of the first phalanges of each side. He moved all the fingers freely, but weakly. The forearms were more wasted than the upper arms. The power was greater at the shoulders and elbows than at the wrists. He had no biceps or triceps reflexes. He had winged scapulæ, but not the typical loose shoulders of myopathy. He had no lordosis and no distinct weakness of the thoracic muscles in breathing. The sensation was normal in all the limbs. Galvanic reaction in the muscles of the front of the left thigh and in the right biceps was about normal, although the AnClC was nearly equal to the CClC, but there was not the slightest slowness in the contraction.

The boy had never whistled. He could blow out a lighted match, but he did not pucker up the lips, nor did he seem able to draw up the corners of the mouth—he simply drew the corners a little backward. He closed the eyes, but not with great force, and yet one would hesitate to say that the orbicularis oculi muscles were weak. The face did not appear to be atrophied. The masseter muscles, the tongue and soft palate were normal, and there was no fibrillary twitching of the tongue. Fibrillary twitching was not noticed anywhere.

MYOTONIA ATROPHICA.

Myotonia atrophica is a rare disease in America so far as one may judge from the scarcity of published cases. The recent critical studies of Batten and Gibb, Ascenzi, Hoffmann, and Curschmann have established its position in relation to the myopathies as a distinct form, and differing in important details from Thomsen's disease.

Batten and Gibb [7] mention that in this disease the myotonic features in many cases are very limited in distribution, and occur after the muscular wasting has been present many years. They admit that muscular atrophy may develop in typical cases of Thomsen's disease. The muscular atrophy, they find, has a distribution which is characteristic, and has been present in fifteen out of twenty-seven recorded cases. There is weakness of the face (myopathic face), atrophy of the sterno-mastoids, atrophy of the vasti muscles of the thighs and dorsoflexors of the feet. The masseter and temporal muscles are sometimes affected, as are also the extensors and flexors of the forearm. The disease tends to affect members of a family in the same generation, and is more frequent in the male sex. The distribution of the atrophy in some cases, especially in the legs, bears a resemblance to that of the distal type of myopathy. Chiefly on these considerations Batten and Gibb believe that myotonia atrophica should be regarded as a form of myopathy.

Ascenzi's reason [2] for accepting myotonia atrophica as an entity are: the great frequency of muscular atrophy and myotonia in the same person. More frequently hypertrophy is found with myotonia (athletic type), but muscular atrophy with myotonia affects symmetrical muscles, and has all the characteristics of myopathy. The atrophy probably has often escaped detection until within a few years. Hypertrophy of muscles may be associated with atrophy in the same case. The clinical picture of myotonia atrophica rests on thirty or more cases, and is fairly constant. The pathological findings are similar in myotonia and myopathy. In both males are more frequently affected, in both symmetrical muscles are involved, the proximal muscles are more affected than the distal, the visceral muscles escape, and other signs of involvement of the nervous system are wanting. Ascenzi gives a table of the reported cases. He states that the type described by Batten and Gibb is the most frequent, but irregularities in distribution of the atrophy occur in cases of this type.

The family reported by Greenfield [29] is remarkable in the number of its members affected with myotonia atrophica and early cataract.

In the two cases reported by Kennedy and Oberndorf [39] the first corresponded to the clinical picture of Batten and Gibb. In the second case there was premature bilateral cataract.

Hoffmann [34] says that all the cases of myotonia atrophica in the literature with his own number seventy, at the most eighty. Of these five had hereditary unilateral or bilateral cataract, and three sporadic, i.e., eight altogether, or 10 per cent. Two or three cases are omitted from this list. The percentage of cases in which two so different conditions occurred is so large that a relation of cause and effect must be considered, especially as usually myotonia exists for months or years before cataract develops. If cataract were observed only in sporadic cases of myotonia, it might be supposed to be caused by the myotonia. Against this view is the fact that there are families in which some members have (1) cataract without myotonia, other members (2) myotonia without cataract, and other members (3) myotonia with cataract. The relation of cause and effect therefore can be determined, and both conditions probably depend upon a hereditary diseased anlage of the organism. The lens is from ectodermic tissue, the muscles are from mesodermic. The fact that persons with myotonia atrophica often are psychopaths is to be explained in the same way. Hoffmann says, so far as he knows, cataract has not been observed in the true Thomsen's disease, myotonia congenita without atrophy. This is true also of other forms of muscular atrophy, dystrophy, neuritis (i.e., the Charcot-Marie type), and spinal forms either acute or chronic. He refers to Lange's cases, but makes no further comment on them, and he does not refer to Hamilton's paper [33]. Lange's and Hamilton's cases make his statement as to cataract occurring only with myotonia atrophica inaccurate.

The recent review of myotonia atrophica by Curschmann [20] with a report of six cases of his own, does much to establish the type he designates by the name of Steinert as a distinct disease.

HYPERTROPHIA MUSCULORUM VERA.

The position of hypertrophia musculorum vera (dystrophia muscularis hyperplastica) is still a matter of dispute. Some of the reported cases have shown implication of the peripheral nerves, but the disease on the whole appears to be primarily muscular, and its place seems to be with the myopathies.

Eichhorst [23] in speaking of this type, says Auerbach believes it is only an early stage of pseudo-hypertrophy of the muscles, but against this view is the absence of proliferation of the interstitial connective tissue of the muscles, even when the condition has existed a long time. Eichhorst describes it as a rare disease, concerning the cause of which very little is known.

Talma [56] in 1893 collected the cases belonging to this type in the literature, and reported a case of his own. His patient was about 48, and had the disorder six years. Almost all the voluntary muscles were very large, and although the panniculus adiposus was well developed, the muscles stood out prominently, so that the man could have served for a model of Hercules, and yet he was weak. A piece of biceps muscle removed during life macroscopically appeared to be normal, and there was an increase of fat or connective tissue. On examination sensation was diminished.

The sarcolemmata had disappeared, the primitive bundles were not sharply defined, and they were farther apart than in normal muscles. Many nuclei were found between the primitive bundles, but it could not be determined whether these were proliferated sarcolemma nuclei or whether they had arisen in the interstitial tissue. The transverse striation had in great part disappeared. Spaces containing spindle-shaped cells were found between the fibrils of the primitive bundles. The muscle appeared to be formed by new primitive bundles, and the number of them was increased. Only those which had preserved the transverse striation were supposed to be capable of normal contraction, and the number of these was small. In strict sense it was not therefore a true muscular hypertrophy, as the muscular tissue was not normal. A piece of nerve examined also was not normal.

In a similar case reported by Auerbach the muscles of only one limb were enlarged, sensory disturbances were not present, and improvement occurred after some months. A contraction of this limb was normal at first, but the power soon diminished. In pieces of muscle removed the transverse striation was normal in almost all the primitive bundles. A slight nuclear proliferation was found. Faradic irritability was diminished.

In Berger's three cases only one limb was affected—objective and subjective sensory disturbances were present, and electrical irritability was diminished. In two of these cases only enlargement of primitive bundles was found.

Friedreich described two cases of true hypertrophy of some muscles associated with atrophy of others. In his first case the muscles of the lower limbs were hypertrophied and very hard in voluntary contraction, and their power was increased. The primitive fibres were thick, and their transverse striation in great part normal, and among these were found wax-like degenerated fibres. In his second case, in almost general atrophy, a few muscles were hypertrophied.

In Krau's case some pain occurred, but no changes were found in the nerves. Hypertrophy of muscles was associated with weakness. Pieces of muscles were excised. Most of the fibres had transverse striation, and nuclei were found within the substance of certain muscle-fibres.

Bruch's case was in a child aged 20 months at the time of death. The muscles of the trunk and limbs were thick and hard, and their power was very great. Microscopically the muscles were normal, but thick.

In Pall's case most of the muscles of the body were enlarged and weak. The primitive bundles were found to be normal in an excised piece of muscle.

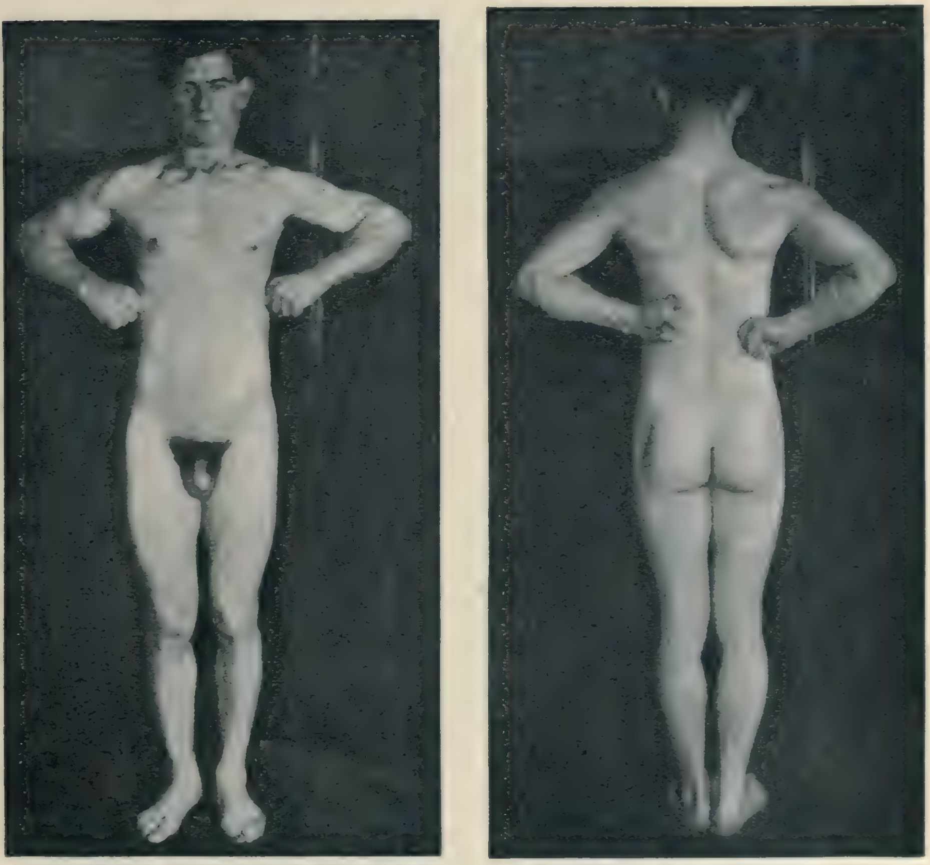
A very interesting case of this excessive muscular development with weakness was observed in our service at the Hospital of the University of Pennsylvania, and was carefully studied and reported by A. H. Woods [59], together with references to the literature. In his summary Woods described the characteristics of the disease as increased bulk of the affected muscles, which feel firm when contracted; diminished power with early fatigue; hypertrophy of individual fibres, some increase in the number of the nuclei; no proliferation of fibrous or fatty tissue, and no degeneration; tendon reflexes, mechanical irritability, and electrical reactions varying from slight diminution to slight increase; no qualitative electrical change; vasomotor changes in a few cases; some dull pain or tenderness of muscles; slow course, with little change from year to year; greater frequency of the disease in males.

In the case reported by Woods, painful muscular cramps occurred, and one of these was seen. A small mass of muscle, 2 to 4 cm. in diameter, rose up, became hard, and formed a rounded tumour, which remained a moment and subsided. Even light tapping of the muscles caused prompt contraction of fibres; in the deltoid and biceps of each side this amounted to a jerk of the whole muscle. These idiomuscular contractions are remarkable, as Babinski and Jarkowski have shown, that while they are absent in myopathy (a fact known previously), they are preserved or exaggerated in neuritis and poliomyelitis. This man was examined in the university gymnasium, and although he was a day-labourer and of unusual muscular development, in muscular power he fell below a man leading a sedentary life, below even the average of 2,000 students, among whom were not merely the young and sedentary, but even some sickly individuals. He fell far below the athletic type of student, with whom he should have compared favourably; the tests being not such as require skill, but those which labourers should best execute.

A piece of muscle was excised from the left triceps and examined. It showed distinct enlargement of fibres in cross-section. The fibres were nearly round in cross-section, the diameters ranging between 70 and 180 microns; most of them falling between 130 and 160 microns; the average being 130 microns. There was no increase in fibrous tissue, endomysium and perimysium being as in normal fibres. There was no sign of fatty overgrowth, and no degeneration of muscular tissue. There was no round cell infiltration.

A typical case of this type has recently come under my observation, for which I am indebted to Dr. W. B. Cadwalader. The patient (figs. 9 and 10) is a man, aged 24. He has six brothers and three sisters, and the brothers appear to the patient to be of unusual muscular development, but without corresponding strength. I have been able to see only one of the brothers, a boy aged 12, who had an attack of poliomyelitis when 19 months old, and has an atrophied right lower limb. He does not appear to be of unusual muscular development. The patient's three sisters are said to be of normal appearance. The man is a plumber, and has done some heavy work. When he was a boy, he says, he was apparently stronger than other boys of his age, but during the past four or five years he has noticed that he is weaker than most men. Four years ago he could carry a bath-tub weighing 280 or 290 lb. upon his back, if some one placed it there, up a flight of stairs. This was difficult to do even at that time, but now it would be impossible. He states that plumbers often carry this weight. He complains of a dull heavy ache in the calves, especially in the morning when he awakens and toward evening. This pain appears to be from use of weak muscles.

When he is stripped he presents the appearance of extraordinary muscular development. His muscles stand out when contracted as do those of a very powerful man. His biceps and deltoid form large masses when the forearms are flexed upon the arms, and the latter are drawn backward. Galvanic reaction is normal in the biceps and deltoid muscles of each side, the KaClC>AnClC, and there is no slowness of contraction. Light touch, pin-prick, moderate degrees of heat and cold are promptly felt in every part of the body. The biceps and triceps reflexes are present on each side, but not very prompt. Either forearm when strongly flexed can be extended by the examiner without much exertion, and with one hand. When either upper limb is held at a right angle with the trunk, it can be depressed by the examiner with slight exertion. There is not the slightest atrophy in any part of the body.



Figs. 9 and 10.—Case of hypertrophia musculorum vera. The muscles are exceedingly well developed, and yet the weakness is very great.

The lower limbs appear well developed, but they are weak. When he attempts to run he almost invariably falls, because of giving way of the lower limbs. He can, however, stand on either lower limb separately. Comparatively little strength on the part of the examiner is required to prevent the man from raising either knee or extending either leg on the thigh. The weakness is noticed especially in climbing stairs. The patellar and Achillis tendon reflexes are about normal on each side.

The man is a Jew. I have been unable to confirm his statement that his brothers are weak, and therefore cannot be certain that the disease is a family one. There can be no doubt that the muscular development is far in excess of the muscular strength. The only sensory symptom is pain, more of a dull ache, although at times it may be more severe; this ache he has always, and has had it since boyhood. I think it is very questionable whether this could be regarded as the result of disease of sensory fibres, and attribute it to constant over-use of weak muscles.

The occurrence of muscular hypertrophy in myotonia atrophica makes the relation of muscular hypertrophy to myopathy more probable. Thus Wardrop Griffith reports a case without necropsy, but with excision of a piece of muscle which presented the symptoms of myotonia in a pronounced form, with marked atrophy of some groups of muscles and an enlargement of those of the calves. In this case there was no increase of the knee-jerks. The histological examination, he says, appears definitely to exclude pseudo-hypertrophic palsy, and to show that there was a real enlargement of the muscular fibres, and that any increase in the general mass of the muscle was not due to fatty or fibrous infiltration. Griffith [30] calls the case one of myotonia atrophica et hypertrophica. The changes in the muscles varied somewhat in the two limbs, upper and lower, but in each instance they were variations in size and in shape of the fibres, and variations in the number and distribution of the nuclei. In diseased muscles the fibres were almost circular in transverse section, and roughly speaking, twice the diameter of those in the control muscle.

A relation of myopathy in some of its types to glandular disturbance possibly may be fully established some day. It is remarkable that in a case of the juvenile type of muscular atrophy reported by J. Michell Clarke [13] there was simultaneous enlargement of the parotid glands. The submaxillary glands were slightly enlarged. This suggests the case of Schönborn, in which all the salivary glands were enlarged, and at the same time there was hypertrophy of all the muscles of the body.

Schönborn [50] thought the case was one of myopathy of toxic nature, possibly from glandular disturbance.

Sacara-Tulbure [48] reported a case of pseudo-hypertrophic paralysis beginning at the age of 48 years, in which she says a remarkable fact was the pronounced development of certain glands, as submaxillary, parotids, axillary, and inguinal. This case was with necropsy.

REFERENCES.

[1] Armand-Delille et Weil, Albert. *Rev. Neurol.*, 1906, t. xiv, p. 190.
[2] Ascenzi. *Monatsschr. f. Psychiat. u. Neurol.*, 1912, Bd. xxxi, S. 201.
[3] Batten. *Quart. Journ. Med.*, 1909-10, vol. iii, p. 313.
[4] *Idem.* *Brain*, 1910-11, vol. xxxiii, p. 433.
[5] *Idem.* *Ibid.*, 1910-11, vol. xxxiii, p. 149.
[6] *Idem.* *Proc. Roy. Soc. Med.*, 1909-10, vol. iii (Neurol. Sect.), p. 92.
[7] Batten and Gibb. *Brain*, 1909, vol. xxxii, p. 187.
[8] Batten and Holmes. *Brain*, 1912, vol. xxxv, p. 38.
[9] Bernhardt. *Neurol. Centralbl.*, 1907, Bd. xxvi, S. 2.
[10] Campbell. *Rev. of Neurol. and Psychiat.*, 1906, vol. iv, p. 192.
[11] Cassirer. Lewandowsky's "Handbuch der Neurologie," Bd. ii, S. 1.
[12] *Idem.* *Ibid.*, Bd. ii, "Specielle Neurologie," Bd. i, S. 230.
[13] Clark. *Brain*, 1903, vol. xxvi, p. 143.
[14] Collier and Wilson. *Brain*, 1908, vol. xxxi, p. 1.
[16] Comby. *Arch. de Méd. des Enf.*, 1906, t. ix, p. 552.
[17] Coombs. *Brit. Med. Journ.*, 1907, vol. i, p. 1414.
[18] Cottin et Naville. *L'Encéphale*, 1912, t. vii, pt. 1, p. 401.
[19] Councilman et Dunn. *Amer. Journ. of Diseases of Children*, 1911, vol. ii, p. 340.
[20] Curschmann. *Deutsche Zeitschr. f. Nervenheilk.*, 1912, Bd. xlv, S. 161.
[21] Dejerine et Thomas. *Rev. Neurol.*, 1904, t. xii, p. 1187.
[22] Duchenne. "De l'Électrisation localisée," third edition, 1872.
[23] Eichhorst. "Handbuch der spez. Pathologie und Therapie innerer Krankheiten," 1907, Bd. iii, S. 932.
[24] Erb. *Münch. med. Wochenschr.*, 1909, Bd. ii, S. 2121.
[25] Eulenburg und Toby Cohn. *Neurol. Centralbl.*, 1911, Bd. xxx, S. 963.
[26] Finkelnburg. *Deutsche Zeitschr. f. Nervenheilk.*, 1908, Bd. xxxv, S. 453.
[27] Gött und Schmidt. *Zeitschr. f. Kinderheilk.*, 1911, Orig. Bd. iii, S. 256.
[28] Gowers. *Brit. Med. Journ.*, 1902, vol. ii, p. 89.
[29] Greenfield. *Rev. of Neurol. and Psychiat.*, 1911, vol. ix, p. 169.
[30] Griffith. *Quart. Journ. Med.*, January, 1911-12, vol. v, p. 229.
[31] Griffith and Spiller. *Amer. Journ. Med. Sci.*, 1911, vol. cxlii, p. 165.
[32] Guillain. *Sem. méd.*, 1907, t. xxvii, p. 277.
[33] Hamilton. *Rev. of Neurol. and Psychiat.*, 1911, vol. ix, p. 645.
[34] Hoffmann. *Arch. f. Ophthal.*, 1912, Bd. lxxxi, S. 512.
[35] Holmes. *Rev. of Neurol. and Psychiat.*, 1908, vol. vi, p. 137.
[36] Holmes, Batten and Barnes. *Proc. Roy. Soc. Med.*, 1908-09, vol. ii (Neurol. Sect.), p. 106.
[37] Howard. *Proc. Roy. Soc. Med.*, 1907-08, vol. i (Path. Sect.), p. 157.
[38] Jendrassik. *Deutsche med. Wochenschr.*, 1909, Bd. xxxv, S. 830.
[39] Kennedy and Oberndorf. *Journ. Amer. Med. Assoc.*, 1911, vol. lvii, p. 1117.
[40] Landouzy et Dejerine. *Rev. de Méd.*, 1886, t. vi, p. 993.
[41] Leclerc. *Gaz. des Hôpitaux*, 1907, t. lxxx, p. 1683.
[42] Lugenbühl. *Deutsche med. Wochenschr.*, 1907, Bd. xxxiii, S. 1439.
[43] Marburg. *Arbeiten a. d. Neurol. Inst. Wien*, 1912, Bd. xix, S. 133.

[44] MERKLEN et SCHAEFFER. *Rev. Neurol.*, 1912, t. xxiii, p. 447.
[45] OPPENHEIM und CASSIRER. *Deutsche Zeitschr. f. Nervenheilk.*, 1897, Bd. x, S. 143.
[46] POLLAK. *Arch. f. Kinderheilk.*, 1910, Bd. liii, S. 373.
[47] ROTHMANN. *Monatsschr. f. Psychiat. u. Neurol.*, 1909, Bd. xxv, S. 161.
[48] SACARA-TULBURE. *Rev. de Méd.*, 1894, t. xiv, pp. 273, 494, 992.
[49] SCHLIVEK. *Arch. of Ped.*, 1910, vol. xxvii, p. 34.
[50] SCHÖNBORN. *Monatsschr. f. Psychiat. u. Neurol.*, 1910, Bd. xxviii, S. 81.
[51] SEVESTRE. *Bull. de la Soc. de Péd. de Paris*, February 21, 1899.
[52] SPILLER. *Med. Rec.*, New York, 1898, vol. liv, p. 9; "Contributions from the William Pepper Laboratory of Clinical Medicine," No. 1.
[53] *Idem.* *Univ. of Penn. Med. Bull.*, 1904-05, vol. xvii, p. 342; *Neurol. Centralbl.*, 1907, Bd. xxvi, S. 508; GRIFFITH and SPILLER, *Amer. Journ. Med. Sci.*, 1911, vol. cxlii, p. 165.
[54] *Idem.* *Journ. of Nerv. and Ment. Dis.*, 1907, vol. xxxiv, p. 14.
[55] *Idem.* "Contributions from the William Pepper Laboratory of Clinical Medicine," 1900.
[56] TALMA. *Deutsche Zeitschr. f. Nervenheilk.*, 1892, Bd. ii, S. 197 (literature to the date of this paper given by him).
[57] THORSPECKEN. *Jahrb. f. Kinderheilk.*, 1912, Bd. lxxvi, S. 300.
[58] WIMMER. *Arch. f. Psychiat.*, 1907, Bd. xlii, S. 960.
[59] WOODS. *Journ. of Nerv. and Ment. Dis.*, 1911, vol. xxxviii, p. 532.
[60] ZATELLI. *Arbeiten a. d. Neurol. Inst. Wien,* 1912, Bd. xix, S. 436.

John Bale, Sons & Danielsson, Ltd., Printers, 83-91, Great Titchfield Street, London, W.

[Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol 40, No. 2, February, 1913.]

SEGMENTAL DISTRIBUTION OF SPINAL ROOT NUCLEUS OF THE TRIGEMINAL NERVE[1]

ANDREW H. WOODS, M.D.,

FORMERLY ASSISTANT NEUROLOGIST TO THE PHILADELPHIA HOSPITAL

For the *spinal nerves* it is believed by many that:

1. Tactile and pressure sensations are conducted through the posterior roots, along the posterior columns of the spinal cord to the *nuclei gracilis* and *cuneatus* of the same side. Thence the fibers form the internal arcuate bundles, decussate and enter the lemniscus of the opposite side.

2. Painful and thermic sensations after entering the cord through the posterior root travel through the posterior horn of gray substance to a column of cells at the base of the posterior horn. New axones arising from these cells cross the median line through the anterior commissure and ascend in the anterolateral tract of the cord (associated with Gowers' tract).

3. The skin areas corresponding to each spinal segment are now fairly well outlined; while the areas corresponding to each peripheral nerve are still more clearly known. It is also well known that no constant relationship obtains between the two, since the area corresponding to any spinal segment may include parts of several peripheral nerve areas. The areas corresponding to spinal segments take the form of bands, like zebra stripes. In the limbs these zones are in the adult longitudinally distorted from the simpler bands of the embryonal limb-buds.

Passing from the trunk to the head, the interesting question is now suggested: What relation obtains between the segments of the nuclei of the *trigeminal nerves* and the skin areas of the face supplied by them?

There is much reason to believe that the trigeminal "spinal root" and nucleus have to do only, or chiefly, with temperature and pain perception. Müller (1) described a case in which a syphilitic lesion destroyed the "spinal root," and the only consequence was loss of pain and temperature perception in the face. In Hun's patient (2) a gross lesion wiped out the "spinal root" and its nucleus, with the result that pain and temperature anesthesia without loss of tactile sense occurred on the same side of the face as the lesion. In this patient the bulbar continuation of the antero-lateral tract (Gowers') was destroyed; thermanesthesia and analgesia (3) of the opposite side of trunk and limbs resulting.

---

[1] From the Department of Neurology and Laboratory of Neuropathology, University of Pennsylvania. Read before the Philadelphia Neurological Society, November 24, 1911.

A number of writers have recently maintained that the proximal end of the nucleus of the "spinal root" receives the pain and temperature fibers of the whole mandibular branch of the trigeminus; the middle portion of the nucleus, the fibers coming from the maxillary branch; while the caudad end of the nucleus is the destination of the fibers bearing painful and thermic sensations from the ophthalmic branch of the nerve. Thus Müller interpreted the case above referred to, as did Spiller (4), a syringomyelic case exhibited before the Philadelphia Neurological Society.

The careful study by Spiller of the course and destination of the fibers of the three branches of the fifth nerve (6) led him to conclude that the bundles composing each branch remain distinct certainly through the Gasserian ganglion. As to their ultimate passage through the "spinal root" and their termination in its nucleus he feels that the arrangement just referred to must be inferred.

The two patients whose symptoms I am about to describe showed sensory disturbances of the face which could not be thus explained; but, on the other hand, strongly suggest a complete analogy of segmental distribution between the *trigemini* and the spinal nerves, so far as pain and temperature conduction is concerned.

Elizabeth E.,[2] a very alert, intelligent and willing subject. Tactile and pressure perception normal over entire body. Pain and temperature perception normal in lower extremities, absent in trunk, except lower abdomen, absent in upper limbs, neck, scalp and parts of the face. On the face pain was felt on the left side anterior to line *4* (see Fig. 1); on the right face anterior to line *3*. Cold was perceived on the left only in front of line *4*, but several months later this area had shrunken so as to be circumscribed within line *2*. On the right cold was felt at the early examinations within line *3*; later,—line *1*. Hot bodies were felt regularly within slightly narrower areas. The patient was examined repeatedly through four months. Her spinal cord at autopsy showed a syringomyelic cavity in each posterior horn, running from end to end, virtually dividing the structure into two

---

[2] The two patients here described were in the Philadelphia Hospital. For the opportunity of studying the former I am indebted to Drs. Burr and Lloyd; for the other patient, to Dr. Charles Potts.

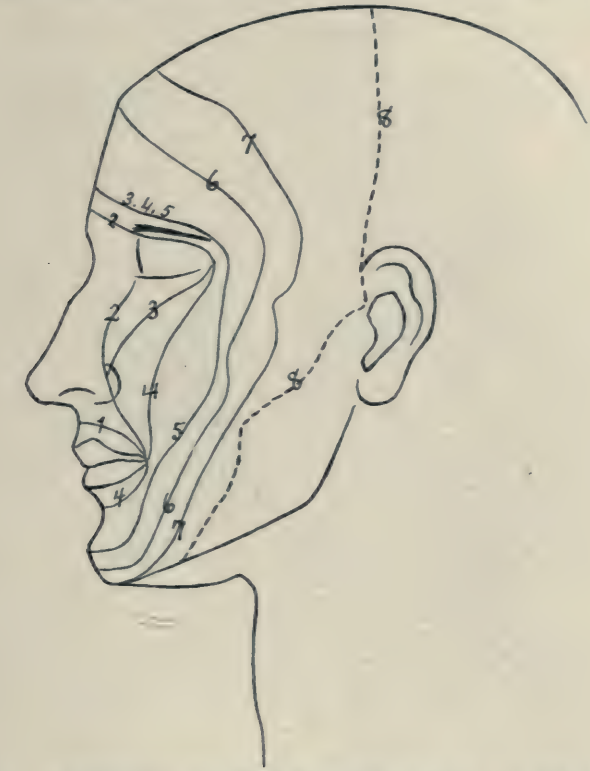


FIG. 1. Temperature and Pain Sensation in Eliz. E— and Chas. B—. *Chas. B—*. *8*, right side: Pain felt in front of this line; *7*, right side: Cold felt in front of this line; *6*, left side: Pain felt in front of this line; *5*, left side: Cold felt in front of this line. *Eliz. E—*. *4*, left side: Cold felt (early) in front of this line; pain felt in front of this line; *3*, right side: Cold felt (early) in front of this line; pain felt in front of this line; *2*, left side: Cold felt (late) in front of this line; *1*, right side: Cold felt (late) in front of this line. *Heat* was felt over slightly narrower zone than cold. *Touch* and *pressure* unaffected. (To facilitate comparison, right and left sides are both drawn upon the one diagram.)

columns, the smaller being composed chiefly of the posterior white columns. The cavities extended into the medulla oblongata, through the gelatinous substance of Rolando almost to the lower limit of the pons. The cavity on the right side extended slightly further cephalad than that on the left. This explains the smaller areas of preserved temperature and pain perception on the right side.

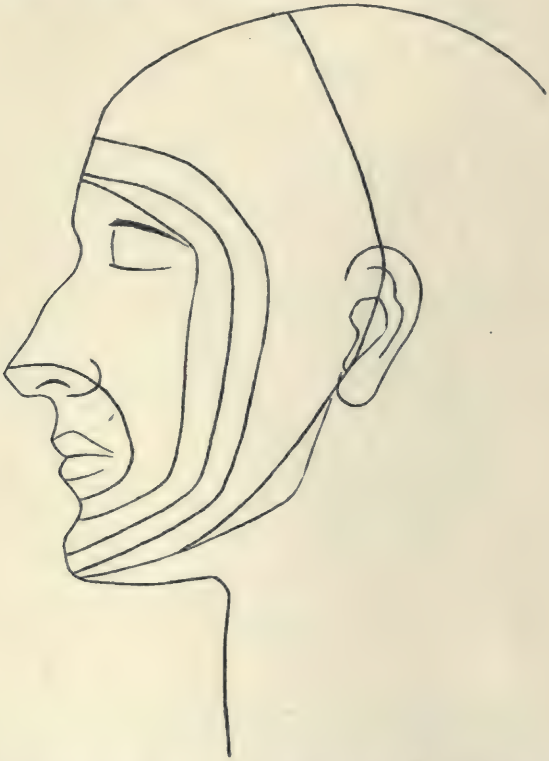


FIG. 2. Kutner and Kramer. Segmentales Fortschreiten der Sensibilitäts-storung am Kopfe. (Lewandowsky, Handb. der Neurologie, Vol. I, p. 787.) See also Schlesinger, Rindfleisch and v. Solder in H. Haenel's article. (Ib., Vol. II, p. 607.)

Stained sections from the lower bulbar region show that each cavity starts dorsally from the bottom of the median sulcus, extends laterally and somewhat ventrally to terminate near the periphery of the section. On the right side the cavity is bridged across by normal structures. At the level of the upper quarter of the olives the cavities extend on each side from the nucleus of ninth and tenth nerves ventrolaterally ventral to and parallel with the intra-bulbar fibers of those nerves to within a short distance of the periphery of the section. The cavities here are more interrupted by bridges of normal tissue. In sections taken from this block a little nearer the junction of pons and medulla oblongata the cavity on the left side has disappeared. In all sections the spinal tract and its nucleus are normal. The cavities extend in such a way as to affect them probably by cutting off their central connections (see Fig. 5). Wallenburg (5) in discussing the central fibers of this nucleus tells of a degenerated tract secondary to lesions of the upper cervical substantia gelatinosa which passed ventral to the twelfth nucleus, decussated and passed upward and somewhat laterally within the formatio reticularis (3). Inasmuch as the cavities in our patient extend ventral to the "spinal roots" of the trigemini and their nuclei, and cut those structures off from the formatio reticularis, it seems certain that the only pathway between the nuclei and the lemnisci is severed. This suffices to account for the sensory impairment in the patient.

The second patient, Charles B., showed no defect in tactile perception. Pain and temperature were perceived above the abdomen in only a few spots, and were not perceived at all on the neck and scalp. But on the right face anterior to line *8* (Fig. 1) pain was felt; while on the left, pain was felt only anterior to line *6*. Distinctions of temperature were correctly made on the right anterior to line *7*; on the left anterior to line *5*. As in the other patient, hot bodies were felt over a slightly narrower area than cold bodies.

Mucous membranes in Elizabeth E.: Corneæ and conjunctivæ very faulty temperature perception; left more often correct than right. Pain was perceived in these membranes poorly; here again the left side showed better perceptive power than the right.

In this woman the oral membranes distinguished painful stimuli and extremes of temperature, but towards the last these perceptions were limited to the tongue and areas near the front of the cavity. The same was true of the nasal membranes, though in them no tests were made of the deep portions.

In Charles B. pain and temperature perceptions were well preserved on cornea and conjunctiva on the right side; but were faulty on the left. In the oral cavity perception of both forms of stimuli was well preserved near the front and on the tongue. Farther back in the mouth, particularly on the cheeks, the various forms of stimuli were confused.

Lewandowsky's "Handbuch der Neurologie" gives diagrams of observations of Kutner and Kramer, and shows the combined results of examinations of the trigeminal sensory areas made by Schlesinger and others (Fig. 2).

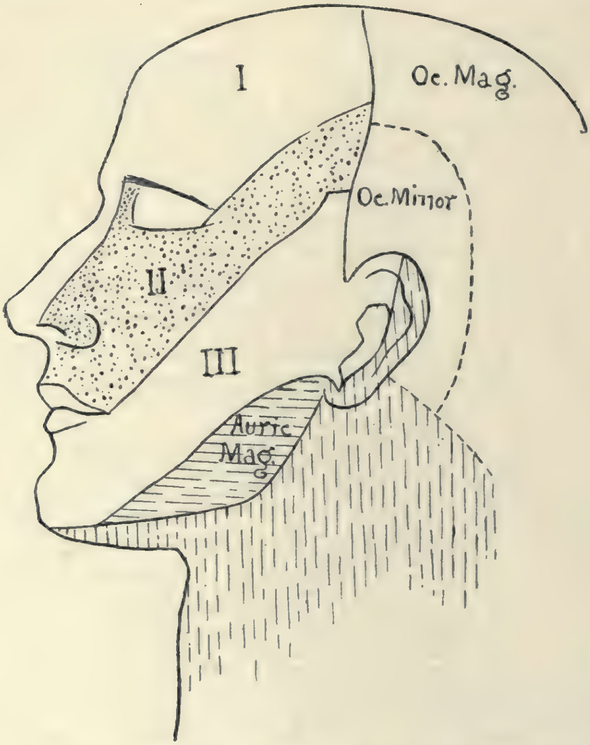


FIG. 3. Sensory skin areas supplied by the three peripheral branches of the V. nerve. After Bing: Gehirn u. Rückenmarks Diagnostik.

The skin areas supplied by the three branches of the fifth nerve are represented in Fig. 3. It is evident that the areas of thermanesthesia and analgesia in our two patients do not conform to the areas to which the peripheral branches are distributed (7). If the fibers of the three branches remain distinct as three separate bundles through the Gasserian ganglion and "spinal root," and terminate in three distinct levels of the "spinal root" nucleus, then no lesion can be imagined which could produce the sensory disturbance here pictured. It is probable that a reassembling of the fibers occurs before they reach the nucleus.

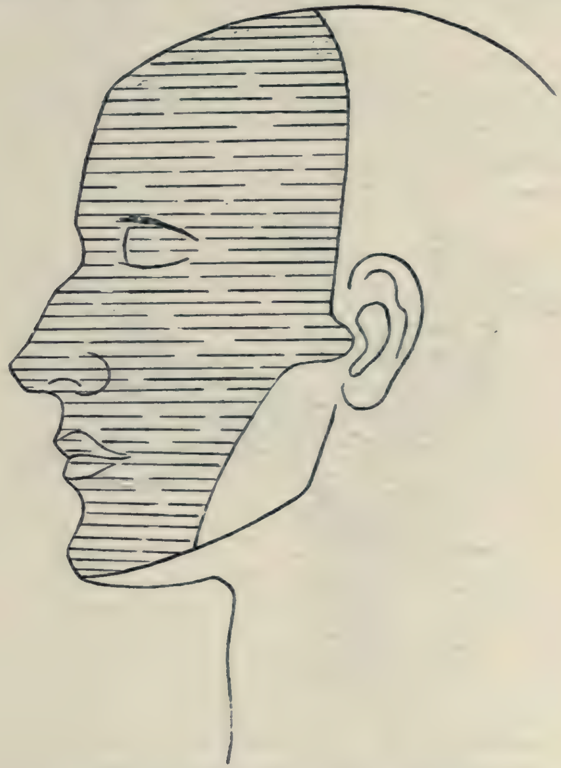


FIG. 4. Average trigeminal sensory area. Cushing. (Howell, Textbook on Physiology, 1909.)

In order to explain the findings in the two patients herein presented I suggest the following as a tentative hypothesis, awaiting confirmation from the study of other cases.

The caudad end of the "spinal root" nucleus receives pain and temperature fibers from a band of skin just anterior to the line shown in Fig. 4, passing from the vertex down to the meatus of the ear, thence to the chin (line *8* in Fig. 1). Some of these fibers come through the first, some through the second, and others through the third, of the peripheral branches of the trigeminal nerve.

Each level of the nucleus (passing cephalad) receives pain and temperature fibers from a concentric band of skin nearer the mouth. Finally the cephalad end of the nucleus receives pain and temperature fibers from the lips and perhaps the tip of the nose.

For the mucous membranes some similar arrangement may exist. Examination of these two patients, however, does not warrant more precise statement.

If a syringomyelic cavity commence in the base of the posterior horn low down in the spinal cord and gradually advance toward the medulla oblongata, it would cut off first the cells already mentioned at the base of the posterior horn which are concerned with thermic and painful sensations. As it reached the upper cervical region in the substantia gelatinosa it would destroy the cells to which these forms of sensation are carried

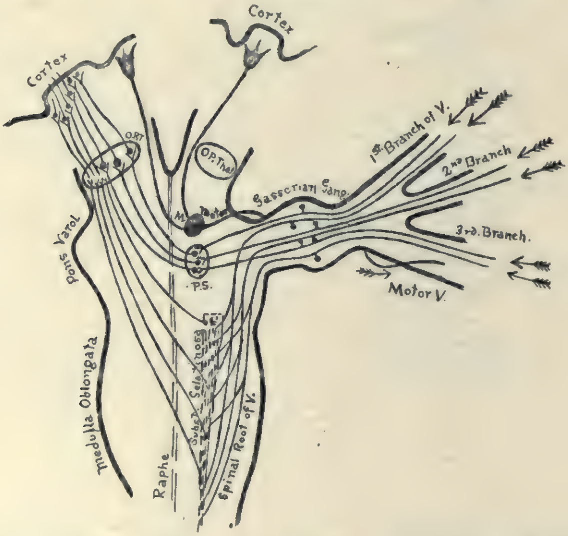


FIG. 5. Schema of fibers and nuclei of trigeminus. Modified from Bing. Tactile and pressure stimuli pass to principal sensory nucleus (*PS*) from all three branches Pain and temperature stimuli reach each level of "spinal root" nucleus through all three branches. Cavity in Eliz. E— cut off, central fibers running from substantia gelatinosa toward optic thalamus; cavity extended from caudad point up nearly to head of this nucleus.

from the two *occipitales* and the *auricularis major* nerves. The next step would involve the lower point of the *nucleus tractus spinalis trigemini*. Thus the sensory disturbance, which had crept up the trunk and neck, would advance upon the scalp and finally involve the forehead and face.

The forms taken by the zones shown in Fig. 1 are better understood if referred to the heads of the lower vertebrates, such as serpents and frogs, in which the somatic axis from snout to tail is parallel with the cerebrospinal axis. The head in these forms is less differentiated from the other (somatic) segments, so that it is not surprising that a segmental distribution of sensory nerves should be found for the head analogous with that found in the trunk. In these animals the final band of skin, corresponding to the most cephalad point of the long temperature-pain nucleus, is around the mouth.

It would thus appear that the trigeminal "spinal root" nucleus may be but a continuation of the pain-temperature nucleus of the spinal nerves.

An apparent difference would then seem to exist between the tactile-pressure arrangement of the spinal nerves and that of the trigemini in that for the spinal segments tactile-pressure fibers are distributed to the same areas to which the pain-temperature set are distributed, while for the trigemini no segmental arrangement of tactile-pressure fibers has been noted. But it must be observed that the skin area corresponding to a given spinal segment has been mapped out by examining the peripheral results of damage to that segment. Such a lesion may affect indeed the nucleus (in the base of the posterior horn) of the temperature-pain fibers; but it affects no *nucleus* of the tactile-pressure mechanism, but only the *bundle of fibers* entering the cord at that level which conduct tactile-pressure impulses. This shows merely that the fibers for all forms of sensation from a particular skin area enter the cord together, those for temperature and pain running at once to a nearby nucleus (in base of posterior horn), while those for touch and pressure run on upward in the posterior columns to the gracile and cuneate nuclei. Later observations may show that the different levels of the gracile and cuneate nuclei are appropriately connected with successive skin areas, corresponding to those of the spinal segments at which the tactile and pressure fibers enter. In the case of the trigemini the tactile-pressure bundle of each nerve is so compact, and the principal nucleus to which it extends is so nearly globular, that any subdivision of either would be difficult to demonstrate.

Therefore it can only be said that for the nuclei of the trunk and head that have to do with touch and pressure perception no segmental arrangement is known. For temperature-pain perception, if these two cases have been properly interpreted, a long nucleus extends on each side from the lower border of the pons down to the end of the spinal cord, related to the substantia gelatinosa and base of the posterior horn, and subdivided segmentally so that each level receives the fibers of a corresponding band of skin. The fibers from any one skin-band whether of head or trunk take the nearest convenient peripheral nerve route, and so may be found in several peripheral nerves; but they ultimately come together again at the proper point in the temperature-pain nucleus.

Why these two forms of sensation should be thus differently transmitted, and why the cells that have to do with temperature-pain perception should be stretched out longitudinally while the touch-pressure groups are so condensed, is another and difficult problem.

CONCLUSIONS

1. It is probable that *touch and pressure* sensations are conducted to the Gasserian ganglion, pass to new axones, enter the sensory root and go directly into the chief trigeminal nucleus. Thence new axones conduct them to higher levels, across the raphe and into the lemniscus. And that

2. Pain and temperature sensations pass from the ganglion, also along secondary axones, go through the sensory root down within the "spinal root" to the nucleus of that root. Thence new axones pass cephalad within the formatio reticularis, decussate and finally reach the lemniscus.

3. The two cases here presented suggest that the facial area connected with the caudad point of the "spinal root" nucleus is a band lying along line *8* (Fig. 1), and that each higher level of the nucleus is connected with a band of skin roughly concentric with the first, but nearer the mouth. Finally a zone of skin around the mouth and nostrils is connected with the cephalad end of the nucleus. The fibers extending from any skin zone to

the corresponding point in the nucleus may pass through all three peripheral branches of the nerve.

1. Müller. Deutsch Zeitsch. f. Nervenk., XXXI, 5 and 6.
2. H. Hun and Van Gieson. N. Y. Med. Jour., LXV, p. 513.
3. A patient of Spiller (Trans. Phil. Neurolog. Soc., Oct. 22, 1909) showed—right trunk and side of head: thermanesthesia with analgesia. Left face: trigeminal tactile anesthesia, nuclear paralysis of V. nerve (motor) of left side. Spiller accounted for symptoms by inferring a lesion in left *tegmentum pontis* which destroyed the antero-lateral (Gowers) bundle with its temperature-pain fibers from right trunk and limbs; destroyed also the recently decussated central fibers from the "spinal root" nucleus of right V. nerve; also destroyed the principal nucleus (tactile-pressure) of left V. nerve with its nearby motor nucleus. (Fig. 5 will make location of lesion clear.)
4. Spiller. See report of case, JOUR. NERV. AND MENT. DIS., Vol. 38, p. 553.
5. Wallenburg. Anat. auz Jena, Bd. XII, s. 95–110. See Barker, "The Nervous System," 1899, p. 646.
6. Spiller. See Review of Neurology and Psychiatry, Vol. VIII, 1910.
7. Bing (Gehirn u. Rückenmarks Diagnostik, under "The Trigeminus") says: "In affections of the most distal part of the substantia gelatinosa in the medulla there follow disturbances of sensation in the frontal region, etc. Thus nuclear trigeminal anesthesias show localizing signs distinguishing them from anesthesias produced by peripheral nerve injuries, just as is true with spinal nerves."

[Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 40,
No. 4, April, 1913.]

PUNCTIFORM HEMORRHAGIC PIGMENTATION OF THE CEREBRAL PIA MATER[1]

BY EDWARD MERCUR WILLIAMS, M.D.

FORMERLY INSTRUCTOR IN NEUROLOGY AND NEUROPATHOLOGY AT THE UNIVERSITY OF PENNSYLVANIA AND ASSISTANT NEUROLOGIST TO THE UNIVERSITY HOSPITAL

In a careful search through the literature, including the well-known works of Mott, Nonne and Oppenheim, I have been unable to find any reference made to what is here described as punctiforme hemorrhagic pigmentation of the pia mater.

Ameuille reported numerous minute hemorrhages in the white substance of the subcortex and the occurrence of small hemorrhagic foci in the pons was described recently by Rimbaud. In a syphilitic case Spiller found hemorrhages in the medulla oblongata and cord in chronic poliomyelitis, and similar extravasations occurred in the acute case reported by Mills.

Large and diffuse subarachnoidal or pial effusions have been mentioned as occurring secondarily to infectious diseases, such as anthrax and scarlet fever, in so-called purpura of the nervous system (Ameuille and Lenoble) and following traumatism or rupture of an aneurysm of one of the basal arteries.

An interesting case of such extravasation involving the pia of a large part of the brain was reported by Leopold. These conditions of diffuse subarachnoidal effusions of blood are all most likely due to a toxic weakening of the blood-vessel walls with subsequent large effusions, but owing to their large size, distribution and distinctly hemorrhagic nature they cannot be confused with the minute areas of pigment such as are present in the specimen here discussed.

---

[1] From the Department of Neurology and the Laboratory of Neuropathology in the University of Pennsylvania.

Obersteiner refers to pigment cells found normally in the pia over the base of the brain and particularly on the anterior surface of the medulla oblongata but pathological extracellular pigment is not mentioned.

The gross specimen of the brain in this condition showed numerous minute brownish pigmented areas of pin-point size.

A microscopical examination of these punctiforme areas showed them to be clumps of granular pigment. They were mostly independent of the blood vessels but the presence in several places of similar pigment granules, though in much less number, immediately around as well as within the vessel walls suggested the probability of their hematogenous origin. Such granules were also scattered in very small numbers throughout the cortical substance but always in close proximity to the vessels. The pigment is for the most part free in the tissue, though some can be found to be intracellular.

The presence at one point in the pia of what looks like an aneurysmal enlargement forming a thickened connective tissue nodule with free pigment and blood cells extending from it into the cortical tissue also shows the blood to be the most evident origin of these areas of pigment.

The luetic infection in this case with its consequent arterial thickening and weakening would doubtless predispose to such condition.

While the pigment would react neither to the test for melanin nor to the one for hemosiderin, it is possible that the material was either too old or the preservative used not a proper one for the obtaining of these reactions. The presence, however, of a faint blue reaction in some of the blood-vessel walls, showing the presence of albuminate of iron, would lend contradiction to such a possibility. The pigment may be in the stage mentioned in the work of Claude and Loyez, in which the ferrocyanide test is not active.

By a careful comparison of the sections with specimens of a melanotic sarcoma, stained by similar methods, there seemed to be a distinct difference between the two pigments.

With the hemalum-fuchsin stain the granules were yellower than the melanin and with artificial light in addition to their being yellower, they showed, by oblique illumination, a glistening metallic, reddish copper colored appearance totally unlike the muddy black color of the melanin. This metallic luster is present, though much less distinct, in sections stained by Weigert's and Nissl's methods, while the melanin sections do not show this high refractive power by any stain. Nissl's stain showed the granules to be dark brownish, those of melanin black. Weigert's gives black color while the color of the melanin pigment is a lighter brownish black.

The general appearance of the pia is that of a case of syphilis. The round cell infiltration is very marked and plasma cells are present in great number. The vessels are thickened and in some of them the adventitial coats are thickened and infiltrated, while in others the overgrowth is confined mainly to the intima.

I am indebted to Dr. Wm. G. Spiller for the privilege of reporting the following case.

G. W., white, age 58, laborer. Admitted to the Philadelphia Hospital February 13, 1907. Family history and patient's early history negative. Present illness began eight days before admission, at which time patient fell from weakness while working but remained conscious. Further history was not obtainable owing to unreliability. The patient denies the use of alcohol, although the first thing he asked for upon awakening in the morning was a "five-cent whiskey." The patient's wife stated that on December 24 the patient was about to go to bed when he suddenly became nauseated, dizzy, and weak in the legs, particularly in the right leg. He fell to the floor, hitting his face, vomited freely and the bowels were evacuated. He was put to bed and remained there one week. Then he got up and seemed well except that at times he was mentally confused. He walked around the house but never returned to work. January 15 he seemed quite bright, but about ten o'clock he had an attack very much like the above and vomiting lasted all night, and till about noon the following day. The patient since then had been mentally confused, though at times he talked rationally, answering questions. He would sleep during most of the day but was very restless during the night, throwing the covers off, etc., but never making much noise. The patient had also been confused at times during the last year.

Examination.—The right lower face seemed paretic—speech was slow and thickened, though the patient said that he had always lisped a little.

The pupils were large and equal and reacted promptly to light and accommodation. There was some ptosis and the left palpebral fissure was wider than the right.

Extraocular movements were normal except upward associated movement which was somewhat impaired.

Tongue deviated slightly to the right on protrusion, and showed some coarse tremor.

Upper extremities were normal in power and movement—no atrophy. Some ataxia, however, was seen in the finger to nose test.

Lower limbs were normal in size and strength, though there was some ataxia by heel to knee test. Knee jerks and Achilles' tendon reflexes exaggerated. A suggestion of ankle clonus was present on the right side. No Babinski on either side.

No vesical disturbance but occasional loss of control of bowel.

Patient's mind was not clear but confused and semi-stuporous. He had to be restrained in bed during the night. He presented the picture of senile confusion with alcoholism.

Dr. Spiller examined him and the notes are as follows:

Patient was decidedly stuporous but he could be partially aroused. Would not do anything he was commanded to do.

The right side of the face was not moved so much as the left during his mumbling. Pin prick on the right side caused him great discomfort and he raised his head and moved the left arm very feebly. The right arm was not moved at all. The biceps and triceps tendon reflexes were normal. He was not aphasic. The left lower limb was moved freely when stuck with a pin; the right only feebly and only at the hip. The patellar reflexes were slightly exaggerated—the Achilles' tendon reflexes were very prompt. The Babinski sign was not obtained on either side.

February 15, '07. The patient had become more stuporous and more restless when awake—struggling a great deal. His nose became reddened and swollen, this swelling extending to the left side of the face.

February 16, '07. Patient had been doing very well—temperature had fallen but in the morning suddenly became very high. Respiration became short, jerky and rapid. There was dullness over the right lung posteriorly, extending anteriorly to anterior axillary line. Numerous râles could be heard over this area. The general condition became gradually weaker in spite of all stimulation, and the patient died at 9 p. m.

For microscopical examination sections were made from the medulla oblongata, and from the cervical, thoracic and lumbar regions of the cord. Hemalum-fuchsin, Nissl's and Weigert's stains were used.

The examination of the cortex shows the pia thickened and intensely infiltrated with round cells and a large number of plasma cells. The vessels are sclerosed, thickened and congested. The ganglion cells are very weakly stained, numbers of them degenerated and the glia may be somewhat increased. Clumps of yellowish pigment as previously described are beneath the pia.

101

In the medulla oblongata the round cell infiltration of the meninges is very marked and the vessels are congested, some of them being so distended and their walls so indistinct that the appearance is that of an intrapial hemorrhage.

Streaks of yellow pigment of irregular sizes and shapes are around some of the blood vessels (hemalum-fuchsin stain)

The cells for the most part are very weakly stained and with a few exceptions, probably degenerated.

In the cervical region the meninges appear thickened, congested and infiltrated and considerable fibrinous thickening is present.

The vessel walls are thickened and many of them hyaline in appearance. Surrounding or close to them are stringy looking masses stained bluish by the Weigert method. The fibers of the anterior and posterior roots are beaded and to a great degree degenerated. In the cord medulla the neuroglia is slightly increased and some of the vessels are congested, but there does not appear to be any degeneration of fibers or tracts of fibers. The ganglion cells are irregularly affected, in some instances staining very poorly, and in many the nuclei are partially disintegrated or entirely gone.

Amyloid bodies are very numerous and show a tendency to linear arrangement along the septæ or the vessels and are particularly thick along the posterior septum. The central canal is large and patulous and the ependymal wall is broken down.

In the sections from the thoracic region the changes are almost the same as in the foregoing. Some of the pial vessels have their walls so disintegrated that the different coats can not be distinguished from the surrounding infiltrated tissue and cells. With the exception of a few fibers to the cells of Clarke's columns none can be found at the entering root zones. The cells of Clarke's columns are degenerated, very few of them having any nuclei and these only indistinct and poorly staining ones. The anterior horn cells are not so numerous as in the normal and some of them are degenerated. In those apparently normal there is an unusual prominence of the protoplasm granules by the acid fuchsin stain.

The central canal is obliterated and the group of ependymal cells seems abnormally large.

In the lumbar segments the findings are about the same with the exception of the presence of a more intense congestion of the capillaries of the gray substance of the cord and a more definite and pronounced overgrowth of the intima in the blood vessels of the pia.

BIBLIOGRAPHY

Ameuille. Bull. et mem. d. soc. d'anat., Paris, 190–.
Ameuille. Soc. med. d. Hop. d. Paris, 1905.
Claude et Loyez. Compt. rend. Soc. d. Biol., Paris, 1911, LXX.
Lenoble. Rev. d. med., 1897.
Leopold. Presented Phila. Neur. Soc., 1910.
Mills. Internat. Clinics, Series XXI., vol. I., 1911.
Obersteiner. Anleitung Beim Studium der Nervösen Zentralorgane, 5 Aufl., 1912.
Rimbaud. Montpell med., 1909, XXVIII.
Spiller. Internat. Clinics, Series XIV., vol. III., 1904.
Spiller. Am. Jour. Med. Scien., 1905.
Vigoroux. Rev. d. psych. Paris, 1909, XIII.

REPRINTED FROM
ANNALS OF SURGERY
227 South Sixth Street, Philadelphia, Penna.
APRIL, 1913.

THE EFFECT OF THE REMOVAL OF THE HYPOPHYSIS IN THE DOG.*

BY JOSHUA EDWIN SWEET, M.D.,

AND

ALFRED REGINALD ALLEN, M.D.,

OF THE UNIVERSITY OF PENNSYLVANIA.

THE problem of the physiology and pathology of the hypophysis represents one of the curious instances in medical science of such contradictory observations, often additionally deflected by speculative fancy, that the answer to any given question concerning the function of the gland in health or disease is even yet not certainly established. The apparently simple proposition—is the organ essential to life?—has divided all workers into two almost evenly balanced groups: those who maintain that the gland is essential to life, and those who hold it to be non-essential.

A detailed review of the literature is here unnecessary, since the appearance of the excellent paper by Aschner (*Pfluger's Archiv.*, 1912, cxlvi, p. 1). Aschner reports extensive experiments made chiefly upon young dogs, discusses the various functions and supposed functions of the hypophysis, and gives a very complete bibliography, covering both the experimental and clinical literature of the subject. We wish in this paper to briefly discuss our own findings, and to leave aside for the present the consideration of results which we have not been able to corroborate.

The work on which this report is based began over a year ago, when the question of the essential character of the hypophysis was perhaps of more importance from the stand-point of experimental medicine than it is to-day. It is evidently a question of prime importance to the surgeon, for if it be really essential to life, the limit of safety in partial removal must be determined. The results in 22 dogs are as follows: 4 died after an average of three days of evident traumatic encephalitis; 1 died of acute lobar pneumonia after two days; 3 died after an average of 23 days of distemper pneumonia; 4 died after an average of 15 days of basal meningitis; 1 died after 38 days, showing at necropsy a thrombosis affecting the hemisphere opposite the field of operation, with a large abscess of the base; 2 died after an average of ten and a half days, the cause of death being uncertain; 2 met accidental deaths after an average of 23½ days; 5 lived for months. None of these dogs showed any immediate symptoms peculiar to the operation, no peculiar gait, nor position, no tremors, nor any other clinical symptom; recovery was prompt and without complications.

---

* Read before the Philadelphia Academy of Surgery, December 2, 1912.

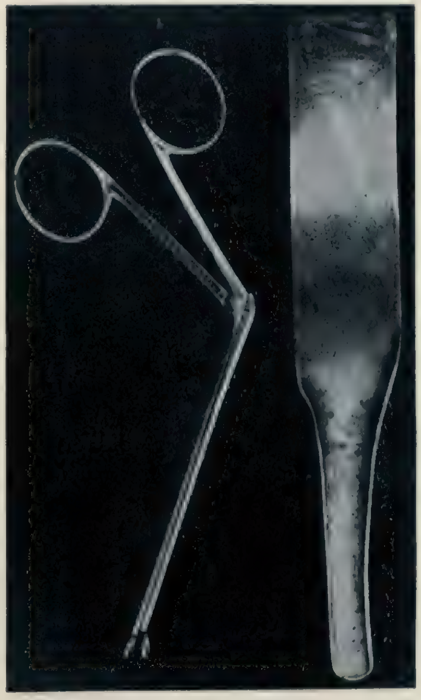
The method of approach is through an incision about 2 inches in length, perpendicularly over the centre of the zygoma—the zygoma forming, as it were, a base line with the two-inch incision extending perpendicularly to such a base line. The zygomatic arch is removed, the coronoid process of the mandible resected, and the base of the skull approached in a direct line. The skull is trephined and the hole somewhat enlarged, and after opening the dura, the brain is carefully elevated by a suitable retractor. The hypophysis is then removed by a special loop forceps (Fig. 1) which enables the operator to grasp the gland and generally remove it in two pieces, the anterior lobe in one piece and the posterior lobe separately. The wound is closed without drainage. With a strong light the field of operation is ample and no difficulty is encountered. This approach, which has been used before, was chosen because the Paulesco-Cushing incision with its extensive removal of the skull seems unnecessary and in our opinion exposes too large an area of the brain to the compressive action of the large masseter muscles. In several instances the operation has failed because of an atypical course of a large branch of the pterygopalatine artery. This branch, ordinarily not in the field of operation, has now and then been unexpectedly cut, with a resulting hemorrhage very difficult of control.

The first point which we sought to determine is whether the entire gland can be removed by this or any other method. Two points must here be recognized: Are we justified in speaking of a complete physiological extirpation when either macroscopically or microscopically some portion of the gland can be recognized after the operation? The anatomy of the gland in the dog is such that it is probably impossible to completely remove all the cells of the pars intermedia. These cells extend for a considerable distance forward toward the chiasm and backward toward the corpora mammillaria. Such a condition makes total removal from a microscopical point of view impossible, but can the gland be physiologically removed? The point is perhaps well illustrated by the history of the work upon the relation of the complete removal of the pancreas to diabetes. It is doubtless impossible to remove completely every cell of the pancreas, and because of this fact the relation of the pancreas to diabetes was for a time in dispute, the explanation being offered that the diabetes was due to the nervous shock incident to the operation. Nevertheless there can be no doubt to-day that enough of the pancreas can be removed to ensure physiological results. It is the same with the parathyroids and with the thyroid, so that a limit of safety has been established by surgeons beyond which it is not safe to remove thyroid tissue. We believe that it is possible to remove the hypophysis physiologically, that is, to remove enough so that certain characteristic changes will follow. Dog No. 1 (Figs. 2 and 3) lived for 38 days, showing no peculiar symptoms until shortly before death, when symptoms of meningeal irritation developed. The autopsy showed an extensive area of softening in the infundibular region, which, as shown in the accompanying plates, must certainly have removed all hypophyseal tissue. Concerning the extent of removal of the hypophysis we have taken the following means of ascertaining whether we have been successful in completely removing it: At the autopsy of several animals which died early, and of all those which lived for months, a block of tissue was removed which included the optic chiasm anteriorly and the corpora mammilaria posteriorly, with about 4 mm. of tissue each side of the medial sagittal plane. This block was mounted in paraffin and serial sections prepared. Careful examination of this material revealed that in only two dogs, dogs Nos. 11 and 22, can it be said that there is no evidence of either pars intermedia or pars anterior. The remnant of the gland found in dog No. 12 was functionally very active, if one may judge by the extensive colloid-like formation and the same appearance of the cells of the pars intermedia which one sees in the normal gland; this dog presents complete atrophy of the testicles.

In the work reported by Aschner with young animals he notes that, in his experience, no marked changes occur after the removal of the gland from the adult dog. The dogs in our series were all full grown. We have no means of judging their exact age except by the fact that they all possessed at the time of operation a complete second set of teeth; in fact, dogs Nos. 19, 20, and 22 were noted as old, their front teeth being worn down almost to the gums.

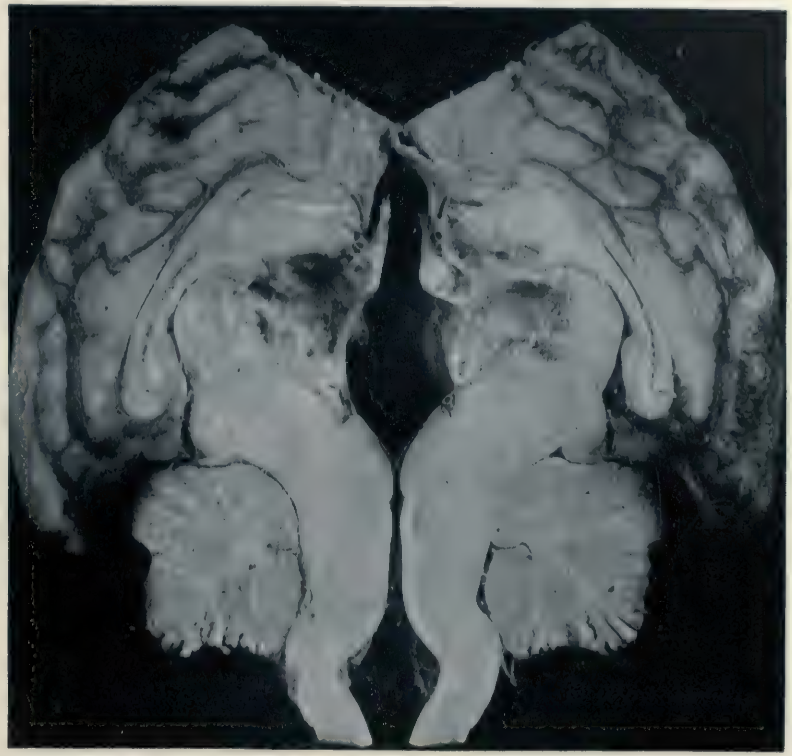
In this series the first change to be noted was an indetermined effect upon the pancreas. At the autopsy of the dogs in the latter part of the series, that is, in those dogs in which we regularly made note of the condition of the pancreas, it is recorded that the organ presented a striking red coloration, having at autopsy the appearance of the gland seen at the height of digestion. Ordinarily at autopsy the pancreas presents the usual picture of a pale, even whitish, organ, the lobules at the edge being rather hard to differentiate from the neighboring fat tissue. In these animals the pancreas was evidently much congested and yet the microscopical study of sections of these organs did not reveal any very marked changes. The changes seen were perhaps identical with those to be found in a pancreas at the height of digestion. The second change in point of time, which we have noted, has been the atrophy of the genital apparatus, particularly of the tes-

FIG. 1.



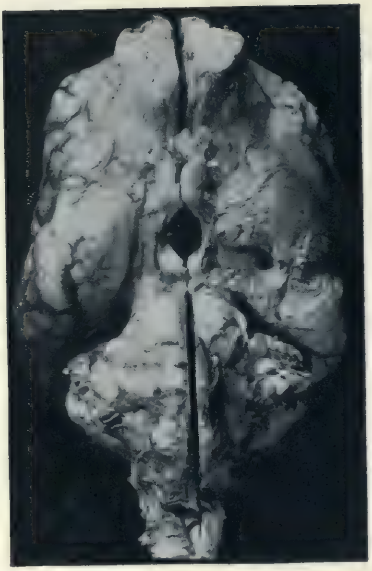
Brain retractor, from beneath, showing rounded edges. Loop forceps, for grasping the hypophysis.

FIG 2.



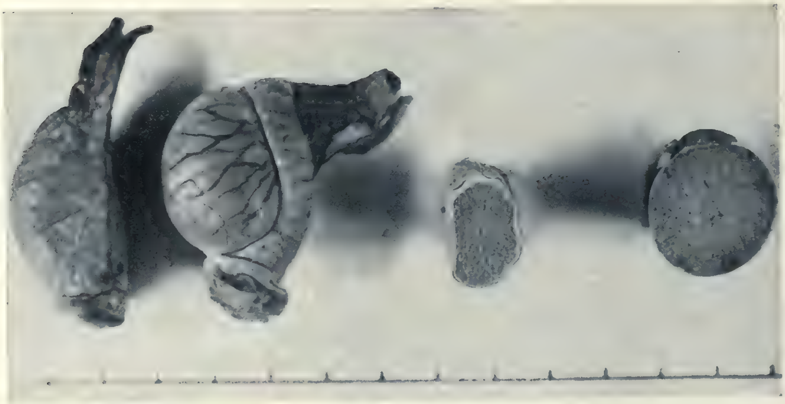
Dog No. 1. Lived 38 days. Shows what must be complete removal of all hypophyseal tissue by the extensive area of softening.

FIG. 3.



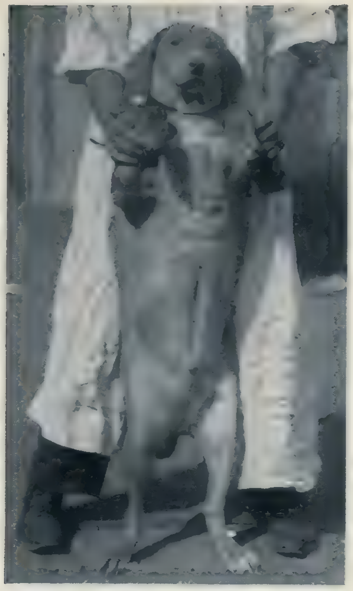
Dog No. 1. Area of softening.

FIG. 4.



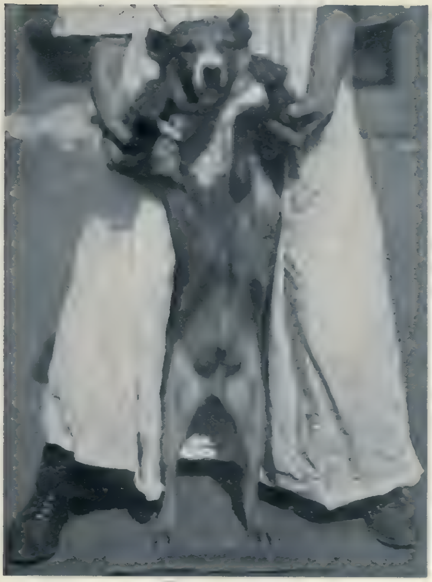
Dog 19. Atrophy of testicles. On the right, testicle removed at time of hypophysectomy. On the left, companion organ, fifteen days later.

FIG. 5.



Dog No. 15. Eight months after operation.

FIG. 6.



Dog No. 13. Eight months after operation.

ticles. From dog No. 19, at the time of the operation (Fig. 4) upon the hypophysis, the left testicle was removed; 15 days later the dog choked to death in an endeavor to swallow a whole cold boiled potato. The autopsy showed that the remaining testicle had undergone a very marked atrophy, due microscopically to a complete loss of the spermatogenetic cells. Dog No. 20, likewise an old dog, showed the same condition of striking atrophy after three weeks. Dog No. 21, dying on the thirteenth day from an undetermined cause, shows no clusters of young spermatozoa in Sertoli cells nor spermatozoa free in the lumen. Spermatids of first and second order are present in moderate quantity. The epididymis is crowded with spermatozoa. Just how soon after the removal of the hypophysis this atrophy commences we are unable to state, but from the three cases cited it is evidently very early. The third change which we have noted has been the increase in weight. This change in our experience does not begin until some time after the operation. For example, Dog No. 15 (Fig. 5), operated on February 7, 1912, weighing about 17 kilos, showed no particular change until the middle of the following June; the dog then weighed 18 kilos. On October 4 the dog weighed 27 kilos. The same may be said of the other dogs in our series (Fig. 6), so that the question has arisen in our minds whether this tendency to obesity is due to a loss of the hypophysis or to a loss of some other function of the body which is controlled by the hypophysis.

The change in the pancreas has been a constant finding; the testicular atrophy apparently is quite constant. Dog No. 16 showed what seemed to be a decided atrophy on palpation some weeks after operation, but at autopsy 7 months after the testicles were normal; this dog showed quite a large rest of pars anterior and pars intermedia, but dog No. 12 showed an equally large rest, with complete atrophy of the testicles, and dog No. 22 showed no demonstrable gland substance, yet had normal testicles. The increase in weight is apparently constant if the dog lives long enough. Perhaps the most important question to-day in connection with the ductless glands is the matter of their interaction; are we to consider the hypophysis, for instance, as a link in a chain, which link, if broken, breaks the chain, or as members of a family, where if one member drops out, he can never be replaced, but the other members of his family can take up his work to a greater or less extent?

In three of the dogs which we have autopsied after several months' time, the thyroid presented a change, the significance of which is hard to interpret. There is an evident increase in the amount of colloid with a flattening of the cells of the alveoli. In summing up our experience with these 22 cases, it is evident that the hypophysis is not essential to life; there are undoubtedly three well-marked changes which follow hypophysectomy; the first change concerns the pancreas. We have not worked very extensively with the question of sugar tolerance and therefore have nothing to say. It is evident that such a study can only be made by determining the individual sugar tolerance before operation and obtaining an index by this means as to the normal carbohydrate tolerance of the individual animal. The second change is the atrophy of the testicles, which is of very early appearance, being extremely marked by the end of the second week after operation. Whether or not this atrophy can be compensated for by the function of some part left behind or of some glandular rests which have been described by Cushing in the floor of the sella turcica, we cannot say. Increase in weight is of late appearance, and whether it be due to the loss of the hypophysis primarily or due to the secondary atrophy of the testicle is not at all clear in our minds.

Our results agree with the most recent work on the subject reported by Aschner, except perhaps in two particulars. He worked almost exclusively with young animals, and inclines to the belief that the removal of the hypophysis from the adult dog is without effect. In the second place he ascribes the atrophy of the testicles to injury of the tuber cinereum. This point of view seems to us a purely academic one. There can be no agreement as to where tuber cinereum ends and infundibulum begins. We have found no evidence whatever in our work which would incline us to agree with him in this particular.

We are presenting this paper at the present time for two reasons: First, we believe that the question of the essential or non-essential nature of the hypophysis is an important surgical matter, and from the results of our work we believe that the entire gland can be removed without danger to life. In the second place, from our work we believe that there is but one surgical indication for operating, namely, intracranial pressure. If such experimental experience is of any value when applied to clinical questions it is our further belief that the intracranial method of approach is to be preferred to any other method, and that the method of operation in human cases which has been elaborated by Frazier is undoubtedly the most correct from the point of view of anatomy and of surgical technic.

Paraplegia in Hodgkin's Disease—Treatment by Laminectomy and the Roentgen Rays.

By CHARLES K. MILLS, M.D.,
Professor of Neurology in the University of Pennsylvania.
And EDWARD MARTIN, M.D.,
John Rhea Barton Professor of Surgery in the University of Pennsylvania, Philadelphia.

(Read in the Section on Surgery of the Medical Society of the State of Pennsylvania,
Scranton Session, September 24, 1912.)

Reprinted from Pennsylvania Medical Journal, March, 1913.

It is worth while to report to a society a single case only when it is unique or presents points of unusual interest. Some study of the literature of Hodgkin's disease indicates the rarity of spinal focal lesions, like the one here recorded, in this affection. Any involvement of the nervous system in the shape of a focal lesion is in fact comparatively rare in Hodgkin's disease, and when present may be the result of invasion of the vertebral canal from without. Advancing disease of the glands of the posterior mediastinum may cause erosion of the spinal column and in this way bring about secondary implication of the nerve roots and cord, with paraplegia and a variety of sensory and reflex phenomena.

One of the very few reports of cases of paraplegia in Hodgkin's disease is that by Warrington. This case of lymphadenoma, terminating in paraplegia, was recorded in the *Medical Press and Circular* (London), Volume 144, Number 23, June 7, 1911. As Warrington remarks in reporting this case, while the lymphadenomatous growths frequently press upon important structures, erosion and the destruction of bone is unusual.

Warrington's patient was a man, forty-six years old, who was admitted to the hospital because of enlarged glands in the neck. The spleen was enlarged and the patient complained of a band of pain round the lower part of the thorax. On examining the excised gland the lymphstroma was found to be greatly reduced and replaced by fibrous tissue. Various examinations with the view of determining the real nature of the case are recorded. The patient left the hospital and returned again in less than a year, wasted, anemic and dropsical. He was then paraplegic to a marked degree and complained of much pain in the legs. The paraplegia rapidly increased until it became complete. Death took place in about four years from the beginning of the illness. Many important advanced lesions of various viscera were found at the necropsy.

The lesion causing the paraplegia was found to be a massive growth that had eroded the bodies of the eighth to the twelfth dorsal vertebræ. This growth had pressed upon and extended downward along the spinal dura. Both macroscopic and microscopic examinations showed the well-known findings of Hodgkin's disease. These and the appearance of the spleen showed that the case was one of lymphadenoma and not a form of sarcoma.

The case which is the particular subject of this contribution is as follows:—

A woman, aged thirty-five, white, single, was admitted to the Hospital of the University of Pennsylvania, April 11, 1912, and was seen by Dr. Mills in consultation with Dr. William S. Newcomet and her brother, a physician. She had suffered several years from what had been designated and treated as Hodgkin's disease.

Four or five days before coming under observation she for the first time noticed some weakness in using her legs. Three or four months previous to this she had had severe pain in the back between the shoulder blades. This in about three weeks disappeared. A week or two previous to her first being seen, the pain had returned and had become gradually of great severity. It was found on examination to be located about the position of the third and fourth thoracic vertebræ. She was seen by Dr. Mills and put to bed on April 7, about one week after the appearance of the paresis in her lower extremities. Four days later she was removed to the University Hospital.

Examination on admission showed that she was suffering from a total paraplegia, the loss of power being complete in both lower extremities. The day after her admission she began to develop paralysis of the bladder and was soon unable to expel urine, necessitating catheterization. The patient had intermittently strong spasmodic contractions of the extremities, these being sufficient to raise the limbs from the bed. At times the legs were drawn up and adducted, and even crossed. At times the jerking was confined to the foot alone and, when the spasms were absent, the limbs had a numb and dead feeling. The abdomen became greatly distended after meals, causing much distress. The patient had profuse night sweats.

The account of this patient before coming under the care of Dr. Mills and Dr. Martin will be given in condensed form in the remarks of Dr. Jopson. It is sufficient here to say that the case at first supposed to be tubercular was later determined by pathological examinations to be Hodgkin's disease. Operations on glandular enlargements in the neck were performed by Dr. Jopson and Röntgen ray treatment was given by Dr. Newcomet with considerable relief of pain and other symptoms.

After admission to the University Hospital several examinations were made both by Dr. Mills and Dr. Spiller, with the assistance of the resident physician, Dr. Manier. The following is a summary of the results of these examinations:—

The paraplegia was complete: persistent but moderate foot clonus was exhibited on the left side, and a slight Babinski response was also obtained on the left.

A complete study of all forms of impairment of sensibility was made—of epicritic, protopathic, deep pressure, tactile discrimination and spot localization. The result of the examinations was to indicate that an intraspinal lesion, at least as high as the fourth thoracic vertebra or higher, was present.

On April 14 she suffered some numbness or peculiar feeling in the little and ring fingers and ulnar side of the left hand and forearm. This was much worse the next day, with increase of pain through the spine between the shoulders. The patient thought this pain was greater than any she had before experienced. It was very severe at night but the next morning the pain was almost gone and the paresthesia in the fingers and hand was much less.

For a day or two the patient had had an increasing sense of constriction about the body at the highest level of impaired sensation, that is about the level of the third rib. At times she felt as if the lower extremities were raised when they were down, or the reverse, indicating loss of the sense of position.

The symptoms continued without much change except some increase in the pain in her back and greater impairment of the sense of position and passive movement in the lower extremities. In some examinations the level of lost and impaired sensibility seemed to be a little higher than above indicated. As the location of a lesion causing impaired sensibility is often somewhat higher than the levels of the nerve roots which appear to be most affected, it was concluded that the upper level of the spinal lesion might be as high as the third or even the second thoracic segment. This was important in deciding the question of the proper location for the laminectomy that was determined upon after consultations. It was concluded that the lamina of the third thoracic vertebra should first be removed, proceeding upward to the second unless the appearances indicated in the field of operation were such as to suggest removing the fourth.

The operation was performed by Dr. Martin. On removing the lamina of the third vertebra a growth was found involving the spinal membranes, and encircling and infiltrating the cord, which was somewhat distorted. The opening was enlarged upward and downward and Dr. Martin removed as much as possible of the growth, but was unable because of its infiltrating character to make this removal complete. The patient made a good surgical recovery. The severe pain from which she had suffered disappeared, the level of the anesthesia descended several inches and the patient recovered some control over the bladder. Improvement in the paralysis was inconsiderable, the patient remaining as before almost completely paraplegic, with exaggeration of the deep reflexes. Röntgen ray treatment was then resorted to again and was continued up to the time of her leaving the hospital several weeks after operation. The applications were made so as to affect the portion of the spinal canal in which the growth was located.

Three months after the operation the patient wrote stating that the x-ray had been continued after leaving the hospital; that her suffering was greatly diminished and that she could sleep through the entire night except because of some disturbance of her bladder.

REMARKS BY JOHN H. JOPSON, M.D., PHILADELPHIA.

The patient first came under my care in February of 1909. At that time she had glandular enlargements on one side of the neck, most marked just above the clavicle, with slight enlargement at the angle of the jaw. At the operation at this time the entire lower portion of the chain of glands was dissected out in the posterior triangle of the neck through a four-inch incision; and another gland was removed through a separate incision at the angle of the jaw. The glands were beginning to show caseation. Sarcoma had been suspected before operation, and our minds were much relieved when this breaking-down process was found to be present.

The question of Hodgkin's disease was not considered at this time. Two competent pathologists reported from different laboratories that the process was tuberculous.

In September, 1909, she was again operated upon for a recurrence on the same side of the neck; the glands removed showed no breaking down. A histological examination was made at this time by Dr. Canby Robinson, now of the Rockefeller Institute, and by Dr. Warfield T. Longcope, both of whom made a diagnosis of Hodgkin's disease. This diagnosis came as a surprise, as the previous diagnosis of tuberculosis had seemed so positive. Shortly before this she had developed a swelling over the sternum, the exact nature of which was a matter of conjecture.

Following this second operation, and from the fall of 1909 down to the time of the development of the paraplegia, this patient had been under observation. During this time she had taken treatment by the x-ray at frequent intervals. She had maintained a condition of fairly good health, although she had suffered at times from pains radiating through the upper part of the chest. She had, however, led a fairly active life and had pursued many of the ordinary pleasures and avocations of her station; but, in April of 1912, it was evident that the pathological process was getting beyond control. There was an enlargement of the axillary glands on the left side. Some glands had enlarged in the neck, and gone down again under x-ray treatment. She was also complaining of more pain. It was about two weeks after this observation that the gait became affected, and paraplegia rapidly developed. The rest of the history has been well given by Dr. Mills and Dr. Martin.

The interesting and unusual points in this case, it seems to me, are: (1) The apparent caseation and softening of the glands, which was noted at the primary operation, and which is not usual in Hodgkin's disease; (2) the involvement of the sternum, probably in the nature of a subperiosteal infiltration; (3) the fact that by thorough early extirpation, combined with subsequent x-ray treatment given over a long period, this patient was held in a fair state of health for more than three years.

If Hodgkin's disease be in the nature of an infection, and the fact that a granular acid-fast bacillus closely allied to the tubercle bacillus has been consistently found in the cases recently studied by Sternberg and others points in this direction, then radical treatment along surgical lines would seem to be warranted. This would consist in thorough extirpation, followed up by prolonged x-ray treatment, and would, of course, be possible only where the disease was localized to one region.

THE DIRECT VENTRO-LATERAL PYRAMIDAL TRACT.

By **WILLIAM G. SPILLER, M.D.,**
Professor of Neuropathology in the University of Pennsylvania.

(With One Plate.)

THIS tract was first described by me at a meeting of the Philadelphia Neurological Society, 28th November 1898. The case in which it was discovered was one of hæmorrhage in the external capsule and lenticular nucleus. The degeneration of the fibres evidently had its origin in this lesion. A little below the exit of the fifth nerve from the pons a band of fibres became separated from the outermost and lateral portion of the pyramidal tract. Lower in the pons this band of fibres passed abruptly backward and entered the trapezoid body. It took a position at the junction of the medulla oblongata and pons lateral to the uppermost portion of the inferior olive. As the olive increased in size the bundle passed backward, and where the olive had its largest width the bundle took a position on the posterior and lateral side of the olive. The bundle was traced below the motor decussation, and remained uncrossed throughout its entire length. It was traced as far as the first cervical segment, but could not be traced further as the spinal cord was not obtained.

The literature on this tract to the year 1913 is given in the work on the anatomy of the central nervous system by Ziehen,[1]

Spiller, *Journal of Nervous and Mental Disease*, 1899, p. 178; *Brain*, 1899, p. 563; *Neurologisches Centralblatt*, 1902, p. 534.

Ziehen, "Anatomie des Centralnervensystems," II. Abteilung, Teil I. (Bardeleben's "Handbuch der Anatomie des Menschen"), 1913, p. 194.

and it would be useless to repeat it. The tract has been observed by numerous investigators. It arises from the pyramidal tract in the pons or medulla oblongata, and when arising in the latter place it is described as passing backward with the anterior external arcuate fibres, or a little interior to these, on the periphery of the medulla oblongata. After reaching the region of Gowers' tract the fibres of the direct ventro-lateral pyramidal tract bend again downward, and pass in this region to the ventral periphery of the lateral column into the thoracic region, or in one case (Bumke) to the lumbar swelling. Ziehen speaks of the tract as the aberrant ventro-lateral pyramidal tract.

In a case I have recently studied the separation of the direct ventro-lateral pyramidal tract occurred in the medulla oblongata, but in a different manner from that described in Ziehen's summary of the literature. The tract did not pass backward with or interior to the anterior external arcuate fibres, but maintained throughout a longitudinal course. The case was one of hæmorrhagic cyst in the right lenticular nucleus and external capsule. The pyramidal tract fibres extended around the lower olive in its upper part, and spread out in a triangular shape immediately posterior to the olive at the periphery of the medulla oblongata. They were all cut transversely in a transverse section of the medulla oblongata, and no rows of black dots were found in the anterior external arcuate fibres indicative of a longitudinal course. The outer portion of the anterior pyramid clearly was interrupted by the development of the olive and the anterior external arcuate fibres. The pyramidal fibres which, in the upper part of the medulla oblongata, extended around the olive were gradually completely divided by the olive and anterior external arcuate fibres, so that a triangular zone posterior to the olive was formed without connection with the anterior pyramid.

The direct ventro-lateral pyramidal tract was spread more along the periphery of the cord at the sixth cervical segment than at the seventh cervical segment; at the latter it was very distinct and more triangular in shape. The direct pyramidal tract on the same side of the cord was much degenerated, but was not continuous with the direct ventro-lateral tract. The crossed pyramidal tract of the opposite side was much degenerated, but there was little or no degeneration in the crossed pyramidal tract of the same side. The direct ventro-lateral tract was still distinct

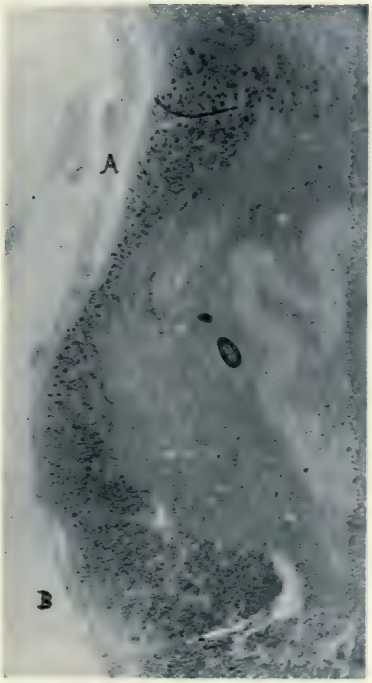


FIG. 1.

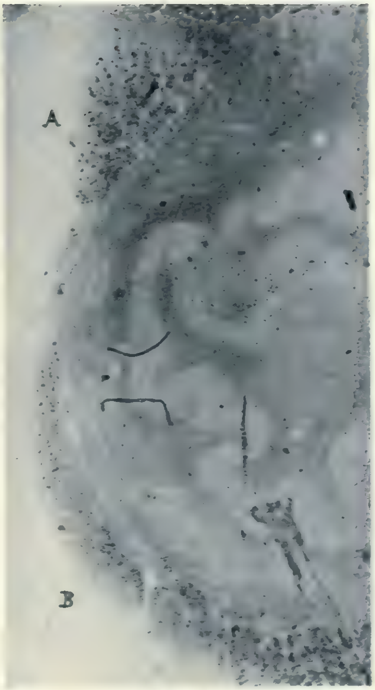


FIG. 2.

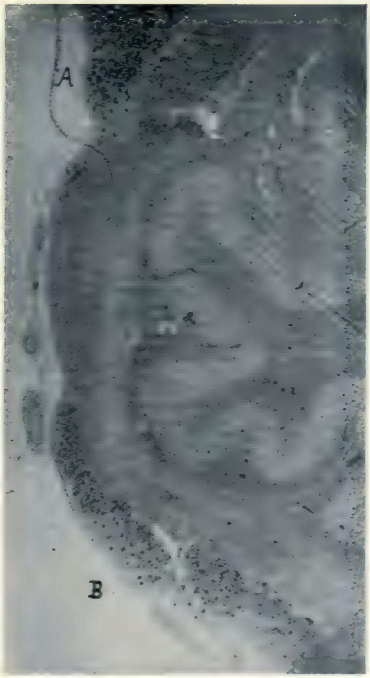


FIG. 3.

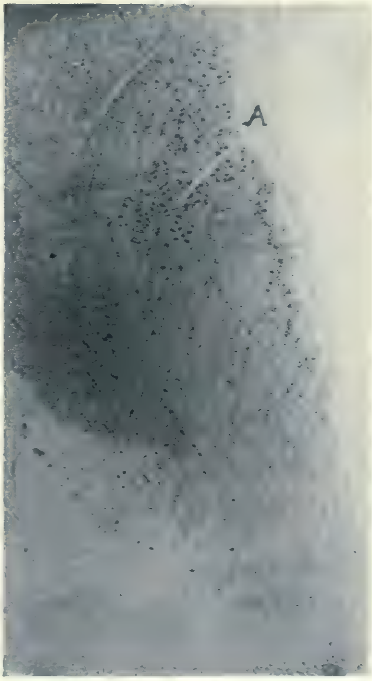


FIG. 4.

at the eighth cervical segment, and of much the same form as at the seventh cervical segment, but was of smaller area. It was not traced below the first thoracic segment.

Description of Figures.

Fig. 1. Showing the fibres of the anterior pyramid, B, extending well around the olive, and united with the direct ventro-lateral pyramidal tract, A.

Fig. 2. The olive and anterior external arcuate fibres are beginning to separate the direct ventro-lateral pyramidal tract, A, from the anterior pyramid, B.

Fig. 3. The separation of the direct ventro-lateral pyramidal tract, A, from the anterior pyramid, B is complete.

Fig. 4. Showing the position of the direct ventro-lateral pyramidal tract, A, in the spinal cord. This section has been reversed in photographing it.

I am indebted to Dr A. J. Smith for the photographs. They have been slightly retouched.

VII.

HISTOLOGICAL CHANGES OF THE CENTRAL NERVOUS SYSTEM PRODUCED BY HELODERMA VENOM.

By Samuel Leopold.

HISTOLOGICAL CHANGES OF THE CENTRAL NERVOUS SYSTEM PRODUCED BY HELODERMA VENOM.

By Samuel Leopold.

The effect of the venom of various snakes upon the cerebro-spinal system has been studied histologically only since 1900. It will not be necessary to review the literature, as this has been done in Noguchi's work on Snake Venoms: An Investigation of Venomous Snakes with Special Reference to the Phenomena of their Venoms (Pub. No. 111, Carnegie Institution of Washington, 1909). It was of interest to compare the action of snake venom with the effect of heloderma venom upon the central nervous system.

Dr. Leo Loeb kindly placed at my disposal the brains and spinal cords of rabbits and guinea-pigs which had been injected with varying doses of the venom of *Heloderma*. The inoculations were either given subcutaneously or into the peritoneal cavity. The dose varied from 25 to 60 mg. Some animals were given but one injection, others two, and in one instance four celloidin capsules had been introduced intraperitoneally and the poison was allowed to act during a period of 28 days. The animals lived from 5 minutes to several weeks. This material for study was placed in 10 per cent formalin or in Muller's fluid, and the sections were stained usually with thionin (Nissl method) and with hemalum. In one animal (rabbit B II, living 13 days), sections of the spinal cord were stained by the Marchi method.

In most cases only the medulla and spinal cord were examined (guinea-pigs 16, 21, 22, 18, A^4; rabbit B^2). In four cases the cerebral cortex cerebellum, pons, medulla, and spinal cord were studied (rabbits Nos. 1, B I, and a^8). In one instance (guinea-pig a^1) sections were taken from every portion of the brain.

For control a healthy guinea-pig's brain was sectioned at various levels from the frontal lobes to the spinal cord, and stained by the Nissl method.

The earliest change noted in our animals occurred after 45 minutes (guinea-pig 18). Some of the cells in the anterior horn of the spinal cord took the stain a little more intensely and diffusely. The medulla and spinal cord of several animals dying at shorter intervals than 45 minutes failed to reveal any changes in their cells. Lamb and Hunter found with the venom of *Naja* definite changes only when the animals lived longer than 2 or 3 hours, but the venom of *Enhydrina* produced pronounced chromatolytic changes in 90 minutes, while Kilvington found chromatolysis with the venom of the tiger-snake if the animal survived the injection of the poison for over 3 hours.

In rabbit No. 1, which lived 75 minutes, some of the ganglion-cells of the spinal cord, medulla, and a few of the Purkinje cells showed an early disintegration of the Nissl bodies. A fine powder was seen around the nucleus, which extended toward the periphery. Some of the cells showed a slight swelling. This latter feature was present in both acute and chronic cases, but seemed a more constant feature in the various sections of the chronic cases. This swelling of the cell does not seem to be a characteristic feature in the pathological findings of others. No mention of it is made by Lamb, Hunter, or Kilvington.

The disintegration of the Nissl bodies was the most constant finding in the acute cases, but it is not characteristic, being also found in the chronic cases.

In none of our acute cases was an eating-out of the cell seen or the complete dissolution of its cytoplasm, etc., as described by Lamb and Hunter. The process after injection of heloderma venom seemed much milder when compared with the effect of some of the snake venoms. In the ganglia-cells of our animals the nuclei were for the most part centrally placed and could always be recognized. They were misplaced only in exceptional cases.

Chromatolysis was a constant feature in the animals living from 48 hours to several days (guinea-pigs A_1, A_4; rabbits B^1 and B^2). In none of the cells was the outline lost; the nuclei were always visible and showed no change in form. These changes corresponded to those of Kilvington, Bailey, Lamb, and Hunter, save that in our cases the cells were in some instances swollen.

Another change which we found and which was not described by the above authors was the presence of minute areas of hemorrhage in two of our cases (rabbit a^3 and guinea-pig A^1). These hemorrhages were small and scattered through the entire structure of the medulla (rabbit a^3), while in guinea-pig A^1 they were noted around the central canal of the cord. These hemorrhages have not been sufficiently constant in our series to conclude that they are characteristic for the venom of heloderma. In guinea-pig A^1 the animal had convulsions prior to its death, and the convulsions may possibly be responsible for the hemorrhages.

We observed a round cell infiltration in two of the chronic cases (rabbits B^2 and A^8); in the former case (B^2) the cell infiltration was found around the posterior septum of the cord, and in the latter animal (a^8) around the blood-vessels. In the cases described by Lamb and Hunter, the cells were clustered around the disappearing ganglion-cells, the process being not unlike a neurophagia similar to that described by McKinas in poliomyelitis; while the infiltration in my cases was purely a chronic inflammatory process. As in the animals poisoned by snake venoms, we also found, after injection of heloderma venom, affections of the various cranial nuclei. In contradistinction to Lamb and Hunter's observations, we could not observe any special selective action of the venom on the third and fifth nuclei; in our entire series no preference could be detected for any one of the nuclei of the cranial nerves.

The changes in the ependymal cells were similar to those seen by previous authors in the case of snake venoms. A vacuolation of the cells was noted and the canal contained an excess of granular substance. In those cases in which hemorrhage occurred in the white substance (guinea-pig A^1 and rabbit a^3) a similar collection of red blood-corpuscles was observed in the canal.

The results obtained with the Marchi method were negative; no changes were found in the spinal cord of the animal living for 13 days. No changes in the myelin or in the axis cylinder were to be noted.

PROTOCOLS.

Guinea-pig No. 16.—25 mg. injected; dead in 8 minutes. Spinal cord and brain placed immediately in 10 per cent formalin. Sections embedded in celloidin. Stained with thionin (Nissl method). Examination of the medulla and spinal cord showed no changes in the ganglion-cells. The vessels were unaltered.

Guinea-pig No. 21.—20 mg. injected; dead in 15 minutes. Brain and spinal cord placed immediately in 10 per cent formalin. Sections embedded in celloidin. Stained with thionin. Examination of sections of the medulla and spinal cord taken at various levels showed no alteration in the cells or blood-vessels.

Guinea-pig No. 22.—20 mg. injected, dead in 30 minutes. Brain and spinal cord placed immediately in 10 per cent formalin. Portions of medulla and spinal cord were embedded in celloidin. Sections were cut at various levels and stained with thionin. Microscopical examination showed no changes in the cells or blood-vessels.

Guinea-pig No. 18.—25 mg. injected, dead in 45 minutes. Brain and spinal cord were placed immediately in 10 per cent formalin. Portions of the tissue were embedded in celloidin, cut, and stained with thionin. Some of the vessels in the medulla were dilated and congested. These vessels lie just beneath the fourth ventricle. Some of the cells of the spinal cord (anterior horn) and medulla (9th, 10th) took the stain a little more intensely.

Rabbit No. 1.—15 c.c. of venom injected; dead in 1 hour 15 minutes. Brain and spinal cord placed immediately in 10 per cent formalin. Portions of tissue were embedded in paraffin. The spinal cord, medulla, and cerebellum and pons were cut longitudinally. Sections were stained with thionin. Slight changes were noted in the cells of the spinal cord (anterior horn cells), the Purkinje cells in the cerebellum, and in a few cells in the nuclei of the cranial nerves. Owing to the manner of cutting the sections the identification of the cranial nuclei could not be assured, but it was evident that the toxin had no selective action, for cells were affected in the various groups of ganglion-cells throughout the medulla and pons. The changes to be noted were an early disintegration of the Nissl bodies into a fine powder, starting around the nucleus and proceeding to the periphery, a migration of some of the nuclei to the periphery, and a slight swelling of the cells. These changes were to be noted in only a few of the cells; the majority were normal.

Rabbit a^3.—25 mg. of venom injected; dead in 2 hours. Tissues hardened and embedded as in rabbit No. 1. The changes in the cerebellum, pons, and medulla were similar to those noted in rabbit No. 1, save that in the medulla numerous small hemorrhages were found.

Guinea-pig A_1.—January 7, 1909, 1 capsule with 0.5 c.c.; January 8, 1909, 5 p. m., dead. Died in 36 hours. Convulsions were noted, together with weakness and paralysis. Tissues and sections prepared as above. Portions were taken for study from several areas of the cerebrum, mid-brain, pons, cerebellum, medulla, and spinal cord. Many sections were cut from these areas. Some were stained with thionin and others with hemalum.

Spinal cord: Sections from the cervical region showed swelling of the cells in the anterior horn, a loss of staining reaction in the Nissl bodies, and displacement of the nuclei. Hemorrhages were to be noted above and below the central canal.

Sections from the thoracic region showed similar changes in the cells and marked congestion of the vessels throughout the white substance.

Medulla: Sections through upper and lower levels. The changes in the ganglion-cells were chromatolysis, swelling of the cells, and displacement of the nuclei. The ganglion-cells of the cranial nuclei seemed equally affected.

Pons: Similar changes were noted in the ganglion-cells of this region.

Mid-brain and basal ganglion: The cells in this region showed a similar picture, though not as many were affected.

Sections studied from various areas of the cerebral cortex showed little change.

Guinea-pig A_4.—24 mg. of venom in capsule. Dead in 48 hours. Sections of the medulla only studied. Quite a few of the ganglion-cells showed loss of staining power, indicative of a chromatolysis. The nuclei of the fifth, seventh, ninth, and tenth were affected alike.

Rabbit B_1.—2 capsules each 30 mg. placed in the peritoneal cavity. Sections fixed and stained as before.

Medulla: Chromatolysis of the Nissl bodies and displacement of the nuclei were seen in some of the ganglion-cells of the cranial nuclei. As these sections were cut longitudinally, the orientation of the various cranial nuclei could not be made out with certainty.

Cerebral cortex: Many of the Betz cells show breaking of the Nissl bodies, displacement of the nuclei, and shrinkage of the cell-body.

Rabbit B^2:—2 capsules, 30 mg. each, in the peritoneal cavity. Killed at the end of 13 days. Sections fixed in formalin and in Muller's fluid.

Spinal-cord sections stained with picric acid showed no changes.

Medulla: Sections stained by the Nissl method show the ganglion-cells of the tenth, ninth, seventh, and fifth cranial nuclei affected. Not all the cells were involved. There was loss of staining power in these cells, although their outlines were well defined. Shadow cells were seen with nuclei faintly present. A slight proliferation of connective tissue was noted in one area around the posterior septum in the medulla.

Rabbit a^8:—4 capsules (1 capsule 50 mg., 3 capsules 0.3 c.c.) given during a period of 28 days. Tissues placed in formalin 3 hours after death. In the medulla the cranial-nerve ganglion-cells showed some loss of staining power of the Nissl bodies. The fifth in this case seemed less involved than the tenth, ninth, and seventh. A few cells showed vacuolation and displacement of the nuclei. The vessels in the medulla were distended and congested. Slight round-cell accumulation was seen in the perivascular areas.

[Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 41, No. 3, March, 1914.]

REMARKS ON THE HISTOPATHOLOGICAL CHANGES IN THE SPINAL CORD DUE TO IMPACT. AN EXPERIMENTAL STUDY[1]

BY ALFRED REGINALD ALLEN, M.D.,

OF PHILADELPHIA

In 1911 I presented a preliminary report before the American Neurological Association entitled "Surgery of Experimental Lesion of Spinal Cord Equivalent to Crush Injury of Fracture Dislocation of the Spinal Column."[2]

I shall briefly recapitulate the essential points in this work because the present report is a continuation of this experimental study.

After laminectomy had been performed on the dog and without opening the dura, there was determined the maximum amount of impact in gram-centimeters to the spinal cord, delivered by a special mechanism, from which the animal would recover with normal motor function, without further operative procedure. Then the amount of impact from which the dog would not recover without operative procedure was determined. The former was termed hypo-impact and the latter, hyper-impact. Any impact up to 340 gram-centimeters was found to be hypo-impact; and anything above this amount was termed hyper-impact.

When an impact of 450 gram-centimeters was used the animal would invariably die, presenting the symptom-complex of complete transverse lesion of the spinal cord unless the operative procedure herein described was followed. This procedure consisted in a median longitudinal incision directly through the injured area of the cord. The physiology of the beneficial action of this means was ascribed to a draining of the cord at this point of blood and serum and thereby not only preventing the pressure of this exudate on the nerve elements but also removing what would in time, through chemical change, give rise to a biochemical irritation with destruction of tissue.

---

[1] From the Department of Neurology and Neuropathology and the John Herr Musser Department of Research Medicine in the University of Pennsylvania, Philadelphia, Pa. Read before the American Neurological Association, Washington, D. C., May 5, 1913, and at the Seventeenth International Congress of Medicine, London, August 6–12, 1913.

[2] Journal of the American Medical Association, Sept. 9, 1911, Vol. LVII, pp. 878–880.

It was pointed out therefore that the question of impact to the spinal cord had two main factors: "(1) the direct injury to the axis cylinders from the impact; (2) the outpouring of serum and blood into the substance of the cord."

Longitudinal incisions through the injured area, not in the median line, but four or five millimeters from the median line and directed centrally, were tried and proved of avail. But theoretically it would seem that the median incision were more free from trauma.

In this presentation I shall discuss (*a*) the histopathological changes in the spinal cord of the dog from fifteen minutes to six hours after hyper-impact injury; (*b*) the effect on the histopathological picture of the median longitudinal incision performed two hours after impact, the dogs being killed six hours after impact; (*c*) consideration of hemolytic change in the extravasated blood in the substance of the cord due to the action of crushed myelin; (*d*) three cases of median longitudinal incision after fracture dislocation of the spinal column in the human subject.

The spinal cord of the dog, fifteen minutes after an impact of 540 gram-centimeters (ninety gram-centimeters more than is necessary to cause an eventual death if not operated on), presents three points of interest. There is, as can be seen in the photo-micrograph, a dense packing of erythrocytes in the pia-arachnoid, most conspicuous on the anterior aspect of the cord. These erythrocytes appear to force themselves into every available cleft and in some places, particularly in the neighborhood of the posterior roots, there seems to be actual continuity between this annular zone of erythrocytes and miliary hemorrhagic conditions in or near the tip of the posterior horn of gray matter. Secondly, there can be seen isolated hemorrhagic foci, usually exceedingly minute and with a predilection to the posterior horns or posterior columns. At times in these fifteen minute specimens the amount of hemorrhage is somewhat greater, but when this is so the bulk of it is confined to the gray matter and apt to be near the central canal. Thirdly, even at this early time there is unmistakably a beginning edematous condition, especially noticeable in the white matter. At first I thought this edematous appearance might be due to a faulty histological technique and I therefore repeated some of this work using several techniques, but I found the same condition of affairs.

One hour after the impact there seems to have been some dissipation in the annular zone of erythrocytes although these are still quite numerous. The most essential change is that the intramedullary hemorrhage has now assumed a much greater proportion. The gray matter is particularly affected in this hemorrhagic condition; and at times there seems to be a dense groundwork of erythrocytes in a large part of the gray substance. Likewise hemorrhages have by this time occurred in the white matter of the posterior and lateral columns and it is an ordinary thing to find places where the walls of the centripetal blood system have been ruptured with a consequent outpouring of erythrocytes. At this time is also noticed a deterioration in the staining characteristics of the anterior cornual cells, a change which is very marked three hours after the impact. Of course this refers only to the centimeter of cord struck.

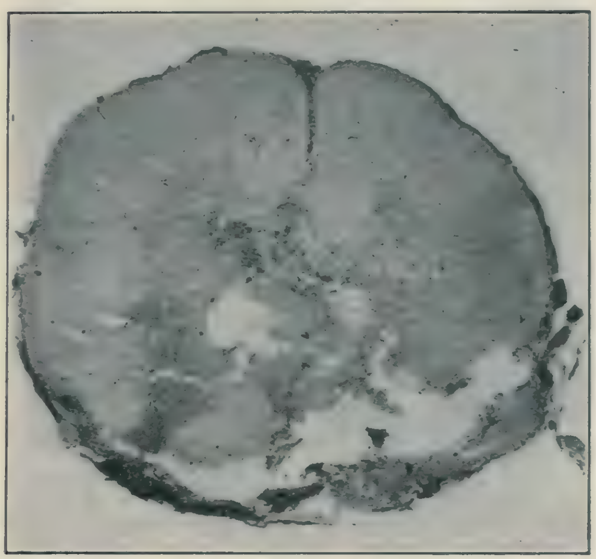


FIG. 1. Cross section of spinal cord six hours after the hyper-impact of 540 gram-centimeters. In this case there was no median longitudinal incision through the injured area. The loss of cord substance is due to the extremely disintegrated condition of the tissue.

Two hours after impact the hemorrhagic areas are conspicuous in gray and white matter, especially the former. There now seems to be but little of the gray matter not infested with erythrocytes and in places this hemorrhage has become hyaline in appearance, showing at the edges of the hyaline areas washed-out ghosts of corpuscles. The edematous condition of the white matter is now quite extreme.

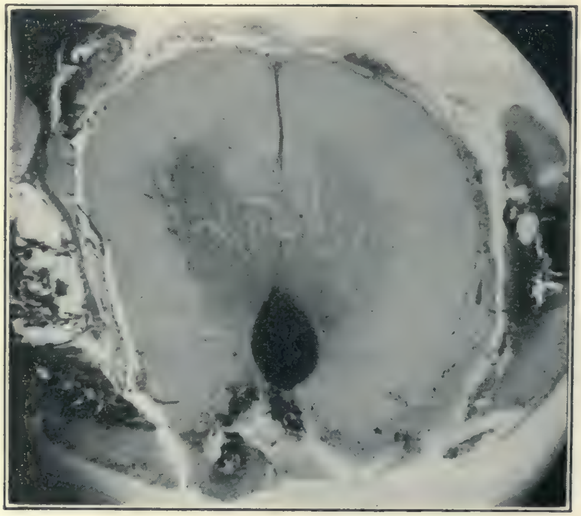


FIG. 2. Cross section of spinal cord six hours after the hyper-impact of 540 gram-centimeters. In this case the operation of median longitudinal incision was performed two hours after the impact. The section shows the blood pouring out of the cord substance posteriorly, into the subdural space. The excellent preservation of the cord substance is quite obvious.

By four hours there has been added one change which surprised me extremely. We find numerous swollen axis-cylinders in the white matter of the lateral and posterior columns. Whether to ascribe this to the first factor (the direct injury to the axis-cylinders from the impact) or to the second (bio-chemical effect of the outpouring of serum and blood into the substance of the cord) I do not feel able to say. But very certain it is that at this stage the swelling of axis-cylinders has commenced.

The fifth and sixth hours after the impact add nothing new to the picture. There is simply an exaggeration of what has already been described above. By the sixth hour there is the very evident disruption of cord substance pictured in the photomicrograph.

I now turn to the subject of median longitudinal section of the cord through the injured area two hours after the 540 gram-centimeter impact. The dog is allowed to live for four hours after this section or six hours after the impact. If the photomicrograph of the cord subjected to the above hyper-impact and removed six hours after injury (*without* the median longitudinal incision) be compared with the picture of the cord having had this operative procedure two hours after the impact and then removed six hours after the impact, the rationale of this means of treatment at once becomes apparent. There is a great outpouring of blood from the injured area posteriorly through the incision as can be seen in the picture. And the most important thing to which I wish to call your attention is the much better state of preservation of the substance of the cord in this sectioned case. We have here decompressed the cord substance.

Noticing the glassy-like appearance of some of the hemorrhagic areas I wondered whether this might be an hemolysis due to the action of injured myeline on the erythrocytes. I therefore made sterile emulsions of dog's spinal cords and tried in vitro to produce hemolysis in whole and defibrinated blood. In this I conspicuously failed. I was the more surprised because most careful examination of these hyaline-like areas under high magnification failed to resolve a granularity.

I shall now briefly note three cases of fracture dislocation of the spinal cord in the human subject in which the median longitudinal incision was used.

The first case was a man fifty years of age who had fallen thirty feet off a scaffolding, landing on his back and head. There was bleeding from the nose and ears as well as complete motor paralysis and anesthesia for all forms of sensation of the lower limbs. The line of anesthesia extended upward to about five centimeters above the umbilicus. The Roentgen ray discovered a lateral displacement of the sixth on the seventh thoracic vertebra. The diagnosis of fracture dislocation of the spinal column complicated by fracture of the base of the skull was subsequently confirmed at necropsy. I saw this patient ten hours after the injury by which time the limit of anesthesia had ascended about fifteen centimeters and his condition was desperate. It was decided to perform laminectomy and view the cord. This was done by Dr. Joseph Spellisy and the cord was found a darker color than normal though of fair contour. The median longitudinal incision was made and there was discharged at once from the cord wound considerable blood.

The next day the limit of anesthesia had begun to fall and in forty-eight hours had fallen to the original level. Unfortunately the cranial condition began to give trouble about five days after the injury and ten days after the accident he died. Dr. Spellisy most kindly gave me the pathological material of this case.

The second case was a large, well-developed negro of about forty years of age who had been struck just below the vertebra prominens by the end of the pole of a rapidly moving wagon. There was a transverse lesion of the first thoracic segment of the cord presenting the symptom-complex of complete solution of continuity. The area of anesthesia ascending slowly and the conditions justifying exploratory laminectomy, Dr. Edward Martin operated about seven hours after the injury.

The course of this case subsequent to operation gave us every reason to hope for a favorable termination. In two days the limit of anesthesia had fallen to the original level and in four days there was a moderate return of deep pressure sense and sense of position *in the lower extremities.* From the first there were indications that only most careful nursing would prevent the formation of bed-sores. His general condition became progressively better and all signs were favorable when, ten days after the operation, his wife insisted on taking him home. He died several days after, his entire sacral region being the site of an enormous bed-sore.

The third case was one of fracture dislocation in the upper thoracic region in a young boy of about seventeen years. Dr. Charles H. Frazier operated four hours after the injury. I learned from Dr. Frazier that when the cord was incised the blood spurted out as if under great pressure. It is now over a year since this patient was operated upon. I have heard that function though not restored is improving gradually.

In conclusion I would say that I feel that this work is still in the experimental stage and that the median longitudinal incision of the spinal cord in cases of fracture dislocation of the spinal column has yet to prove its position as an advisable procedure in surgery. There are two points which I would accentuate. The first is that it seems as though it would be necessary in order to have this method effective to operate within a very few hours of the injury. This is without doubt a great limitation. Secondly, one will always be able to say that possibly a given case would have recovered if left alone; or that the semisuccessful outcome of the operated case is possibly owing to additional trauma to the cord at the operation, and that if the expectant treatment had been followed the results had been better. Of course this latter argument can be used with just as much force in the opposite direction. But let us right here remember that the usual course of the broken back is death after much suffering and that in the few cases that live after the expectant treatment in the vast majority the last state of that man is worse than the first.

The results from the experiments on the dog are most encouraging and this encouragement is more than sustained by the few observations in the three human cases.

THE MENTAL MANIFESTATIONS OF EPILEPSY

BY

N. S. YAWGER, M.D.
PHILADELPHIA
Examining Physician to the Pennsylvania
Epileptic Hospital and Colony Farm

◆

REPRINTED FROM

THE JOURNAL OF ABNORMAL
PSYCHOLOGY
BOSTON

FEBRUARY-MARCH, 1913

THE MENTAL MANIFESTATIONS OF EPILEPSY[1]

BY N. S. YAWGER, M.D., PHILADELPHIA

Examining Physician to the Pennsylvania Epileptic Hospital and Colony Farm

OF all known diseases, none have such mysterious and weird manifestations as epilepsy. In some unexplained way the process of sleep may be the only assignable cause of an epileptic attack, and this constitutes what is spoken of clinically as the nocturnal form of this disease: if one subject to night seizures sleeps during the day, the attacks may occur then, and it is often during the deepest sleep that a fit is provoked; when there is but an occasional nocturnal convulsion, the malady may exist throughout life and remain unrecognized; and, when the seizures are mild and infrequent, their occurrence in some persons has been effectually concealed for a long time. One afflicted with this disease may, without a moment's warning, be felled to the ground in an unconscious and spasmodic state; sometimes a seizure does not cause more than a fleeting and an almost imperceptible change in the psychic equilibrium; or, again, the manifestation may be so grave as to instantly drive its victim to the utmost violence, and subside, leaving him entirely oblivious of his actions and deeds.

Of recent years the mental manifestations of epilepsy have received more and more serious consideration. Previously, the motor disturbance was regarded as the cardinal element in the disease, but most authorities now believe that in order to establish the diagnosis of epilepsy there must at least be some attacks in which there is sudden, temporary impairment or loss of consciousness.

Some writers have endeavored to establish a relationship between genius and epilepsy, based upon the statement that a number of great men have had, or were alleged to have had, this disease. Among those cited are Mohammed, Cæsar, St. Paul, Mozart, Richelieu, Swift, Swedenborg, and Napoleon; we have no good reason for believing that most of these men had epilepsy, and there probably is no such relationship; as all segregations of epileptics will show, there are enormous numbers who at no time during their existence have evinced more than commonplace mentality; indeed, intellectual impairment is to be expected in these individuals, and the instances where complete mentality has been preserved are most exceptional. Undoubtedly, it is true that disease amounting to an affliction has at times kept individuals away from the less serious things in life, and for this reason they have abandoned themselves to hard work and their endeavors have led to fame; but great men afflicted with a disease of an epileptic nature, undoubtedly, have not become famous because of this association.

---

[1] Read before the Chester County Medical Society at Embreeville, Pa., July 9, 1912.

From the Department of Neurology in the University of Pennsylvania.

Since epileptic seizures are prone to have a disintegrating effect upon the mentality of the individual, these insults, when often repeated, almost invariably leave a permanent impression upon the mental state of its victim; this, with the terrible nature of his malady and its probable incurability, together with his being set apart from normal persons and conditions, have been the causes given for the development of what is termed the epileptic character and temperament. "It consists," Peterson[1] states, "of a mixture of melancholy, hypochondriasis, emotional irritability, moroseness, distrust, misanthropy, mental apathy, and dullness, often combined with religious tendencies and modified by pathological psychic conditions incident to the ravages of the disease." However, it may be that the development of this state, so peculiar to an epileptic, is due in part to the mismanagement of these unfortunate persons by their families, and not to such a marked degree by the disease itself; this seems more especially true of that class of cases where there is not sufficient means to provide suitable attendants for these children; and this point is rather strikingly brought out in "The Autobiography of an Epileptic."[2] This individual, when a child, was unusually bright; he had his first attack in school, and he was not allowed to go there longer; later, while out with one of his family, he had an attack on the street, and after this he was kept at home most of the time; he was accused of violence towards the younger children; of this he knew nothing, and he believed such stories were told against him for spite. Therefore, having no knowledge of these misdoings, he naturally thought that instead of lying, he was lied about. This epileptic writes: "The injustice I suffered, or thought that I suffered, made me morose, jealous, suspicious, brooding, and I could hardly be civil to those belonging to my home. I despised them all and felt that I should like to do the things they accused me of doing." At times it may be as one writer says, "The epileptic is forced to develop to some extent on the lines of the untutored savage. This fact explains why the full-grown epileptic has often many of the qualities of the savage."

Epileptic seizures do not always appear unannounced, as an aura or warning frequently precedes the attack; this may occur alone or in association with a warning in another part of the body; sometimes such manifestations consist of the subjunctive sensations of smell and taste; or the individual may feel intensely apprehensive, and, as the instinct of fright is often the subjunctive feeling or emotion of fear, a patient in this mental state may be impelled from home. One epileptic whom I recall seeing suddenly bounded forward directly towards a large tree, and had he not been caught by an attendant and brought to the ground he would probably have been injured; his fit followed in a few seconds. If a psychic aura were the only manifestation of an incomplete attack, the absence of motor symptoms would render the disturbance indistinguishable from psychic epilepsy. Hughlings Jackson[3] wrote of a "dreamy state" in association with what he termed an intellectual aura, and this mental condition a patient of his described by stating that "he felt as if he were saying, doing, and looking at things which he had experienced before." This, it seems, amounts to a psychasthenic state occurring in an epileptic, and for this condition Janet[4] has coined the term "psycholepsy."

As previously stated, some disturbance of consciousness is of paramount importance, and it is now believed by many that to establish the existence of epilepsy there must be sudden, temporary impairment or loss of consciousness; it is true at times phenomena are observed in these patients in which there is no such disturbance, but to establish the diagnosis there should be at least some attacks in which the cardinal symptom, a sudden temporary disturbance of consciousness, is actually present. Such disturbance may be slight and momentary, or it may amount to absolute unconsciousness and exist for several minutes; again, it may be of several months' duration; sometimes, during such a period, the patient acts most strangely and usually without subsequent knowledge of his behavior. With this disturbance of consciousness as the requisite, one cannot but feel that a diagnosis of epilepsy may not be made if the attacks occur only during sleep.

Just how such disturbances of consciousness are brought about it is impossible to positively state, but the best explanation appears to be that of discontinuity of conduction at the junctions of the neurons.

In a complete epileptic seizure, absolute unconsciousness usually follows promptly after the appearance of the aura if this is present; it continues through the tonic and clonic stages and into the period of stertor. This unconsciousness usually lasts from a few seconds to about five minutes, and is followed by a period of perhaps half an hour, during which time the patient lies profoundly comatose; natural sleep then gradually supervenes, and from this the individual awakens somewhat confused; this confusion may be brief or it may persist for hours. If seen only in the stage of coma, the possibility of the condition being due to alcoholic or other drug intoxication, apoplexy, diabetes, or hysteria might have to be considered.

In status epilepticus fit follows fit, at times even to several hundred, and without the return of consciousness; this is the gravest of epileptic paroxysms, and it leads to about ten per cent of the deaths occurring among epileptics. The status may be precipitated by some such cause as a blow, it may be induced by an acute infectious process, it is at times the termination of long standing cases of epilepsy, and frequently it develops without assignable cause. So-called serial epilepsy may be regarded as a subacute status.

Complete temporary loss of consciousness was observed in a case which I examined recently; while making a neurologic study of this patient, I observed that she drew rather a long inspiration, and upon glancing at her face saw that there was just a slight suggestion of tremor about her eyelids; her eyeballs turned upward, and she wavered slightly, but she did not fall; in about ten seconds she looked at me in a dazed sort of a way and then regained complete consciousness. Upon two occasions I chanced to have a sphigmomanometer attached, and during the seizures there was no appreciable fall in blood pressure. This patient had no previous warning of these attacks, nor had she subsequent knowledge of them. Seizures of this kind are classed as psychic epilepsy; they are not uncommon, and at times pass for cardiac syncope. Such manifestations lend additional gloom to the prognosis. Some persons are aware of having had such attacks, and it has been my experience while examining patients to have them tell me that they have just had a "spell," and these may be so mild and of such short duration as to be unobserved except by the patient. Sometimes major epilepsy has begun in this way, and it may then be several years before the more severe symptoms appear.

It is important and at times difficult to distinguish between psychic epilepsy and cardiac syncope; in the latter condition the disturbance of consciousness is less sudden and less complete, except, of course, when the attack is fatal; if the individual falls, there is scarcely ever an injury sustained; mental confusion does not follow; the reflexes are less apt to be abolished; pallor of the face is observed, and in psychic epilepsy this probably is not the first sign; weakening of the pulse is present in syncope, but it is scarcely ever an accompaniment of epilepsy; the patient may attempt to rise and again lose consciousness; if a sphigmomanometer observation is possible, a fall of blood pressure is shown.

Strange to say, epileptic attacks may be for a time inhibited by anything that holds the attention of these patients. It has often been observed at the Oakbourne Colony, that during an entertainment of an hour's duration, the children, perhaps, are all free from seizures, whereas, during the same period at another time, five or six fits would probably occur. It is also well known that an acute illness, either infectious or otherwise, usually has an inhibiting influence upon epileptic seizures. The attacks may even cease for months, but they almost invariably return. The cause of this strange influence is not known.

The mental manifestations of epilepsy have been comprehensively classified by Turner[5] who considers the subject under the following headings: 1. Epileptic temperament; this has already been mentioned. 2. Paroxysmal psychoses, which either precede or succeed the convulsive phenomena. 3. Paroxysmal psychoses, which replace single fits or a series of fits (psychic equivalents). 4. Permanent inter-paroxysmal mental condition. Some of these headings are extensively elaborated by this writer.

About ten per cent of all epileptics become insane, and these constitute some ten per cent of the population of asylums. Since the disease in itself is seldom fatal, epileptics usually living beyond middle life, these patients "accumulate" and, therefore, they do not represent ten per cent of the admissions into asylums. However, these figures are affected by the increasing number of patients maintained in hospitals and in colonies devoted to their exclusive care. In all places where large numbers of epileptics are segregated, a few are found to be the victims of moral depravity.

The paroxysmal psychoses may precede, may succeed, or may replace the convulsive phenomena; the last are known as psychic equivalents.

Epileptic insanities are of various types; they may be broadly included under the term manic-depressive manifestations, or they may be classed separately as mental confusion, hallucinosis, mania, melancholia, and acute dementia; however there is this distinction to be made from the classic psychoses — acute epileptic insanity is more precipitate, more intense, of shorter duration, and terminates more abruptly than insanity of other origin.

The epileptics in insane hospitals are invariably classes as the most dangerous and the most troublesome patients that require detention.

Preparoxysmal manifestations sometimes present themselves, and they are important as their recognition enables those in attendance to be watchful of these individuals.

Such symptoms are known as prodromes, and they consist for the most part of irritability, excitability, depression, unusual slowness of cerebration, insomnia, a feeling of unrealty, and occasionally hallucinations; these manifestations may occur hours or even days before the attack; while in this state the patient is sometimes impelled from home; the prodromal symptoms mentioned are not to be confounded with the psychic aura previously described; the latter is brief, and it invariably occurs immediately before an attack, with this possible exception, that, rarely, the attack may fail to appear.

An epileptic psychosis most commonly succeeds the convulsion, and it usually lasts but a few hours, although in rare instances it may extend over a period of weeks. The milder manifestations are for the most part delirium, stupor, or confusion, but the rapidity of the actions and thoughts may conform to the more serious condition of mania, and acts of violence are at times committed; there is usually no subsequent recollection of the conduct during such a period, but at times the individual may have a hazy idea of what has happened. A patient whom I have seen on a number of occasions, forty years old, had convulsions in infancy, has had minor epilepsy for the last five years, and major attacks, occurring about once a month, for the last three years. During the interparoxysmal period his conduct is exemplary, even better than that of the other forty-eight patients in the same ward; but for about ten minutes following a fit he often becomes dangerous, and he may then attack any one who comes near him; once he succeeded in getting a knife and concealing it under his pillow; because of these brief dangerous periods he is of necessity detained in an insane hospital; all subsequent knowledge of his actions during the periods of excitement is denied by this patient. The sudden deprivation of large doses of bromide may at once cause an acute psychosis to develop.

Automatic acts are very frequently to be observed after epileptic seizures; perhaps the most common of these is an attempt to undress, and Ernest Jones[6] is probably in error when in this connection he says, "The tendency to undress partly that is so frequently seen just after an attack of major or minor epilepsy is also of exhibitionistic origin." In refutation of this, I know of patients who automatically assume the attitude of prayer almost as often as they attempt to undress; furthermore, I have questioned female attendants of large experience among male epileptics, and have been informed that during automatic acts they have not observed anything suggestive of indecency; probably, as Gowers[7] suggests, this tendency to undress is from some vague sense of indisposition and the propriety of going to bed. I recall one patient who would at times undress, place on her night garment, and actually get in bed before returning to complete consciousness.

The more extensive phenomenon of automatism is occasionally observed, and this may become a question of the gravest medico-legal import; in this mental state the patient sometimes behaves most strangely; occasionally he may expose his person, or perhaps he may wander from home and be gone for weeks, or even months; overt acts are at times committed, and upon the return of complete consciousness there may be no knowledge of the behavior during such a period. If this state were to appear independently of any motor or sensory disturbance, it would constitute the psychic equivalent of epilepsy. As Spratling[8] says, "Psychic epileptics may commit all manner of crime; theft, arson, rape, assaults, homicides. They are not infrequently pyromaniacs entirely without reason or impelled by the flimsiest motives." It should be borne in mind that occasionally automatism is met with also in alcoholism, hysteria, and in degenerate persons.

Confirmed epileptics are much inclined to religious observances; this, however, does not appear to arise from any deep-seated religious feeling, but rather as an automatic process resulting from previous religious training; sometimes marked religious enthusiasm precedes a maniacal outbreak. Spontaneous attacks of crying or laughing may appear.

Dream states, or a stuporous condition, may develop and sometimes there is a maniacal episode; also, narcolepsy has been observed to replace attacks, and from this the patient cannot be awakened.

Somnambulism, which is really the acting of a dream, occasionally occurs among epileptics.

Generally speaking, after the disease has persisted for some time, the memory becomes impaired and this amnesia follows the usual rule where memory fails gradually, in being most defective for recent events. Since the failure of memory cannot be reproduced, it is an amnesia of conservation, and not, as Ernest Jones[6] has said, an amnesia of reproduction; in the latter instance the failure of memory might be recalled by hypnosis, and the state should then be regarded as one of hysteria.

Mental failure need not be the necessary result of epilepsy, as in rare instances the affection has been observed, not to be incompatible, with retention of all the normal mental faculties. Usually, however, there is an ultimate dementia, and it differs in nowise from that of other insanities except by the presence of fits. Epileptics do not often attain to an extreme age, but they may live sufficiently long for the dementia to become absolute.

References

1. Church-Peterson. Nervous and Mental Diseases. W. B. Saunders Company, Philadelphia, 1908; p. 812.

2. Alexander. A Clinical Lecture on the Autobiography of an Epileptic. The Medical Press and Circular, April 12, 1911; Vol. XCL., p. 382.

3. Case of Epilepsy with Tasting Movements and "Dreamy State," Brain, 1898; Vol. XXI, p. 580. Epileptic Attacks with a Warning of a Crude Sensation of Smell and with an Intellectual Aura (Dreamy State); Brain, 1899; Vol. XXII, p. 534.

4. Les Obsessions et la Psychasthenie; Felix Alcan, Éditeur. Ancienne Librairie Germer Baillière et Cie; Paris, 1903; Vol. I, p. 502.

5. Epilepsy. Macmillan & Co., London, 1907; p. 118.

6. The Mental Characteristics of Chronic Epilepsy. Maryland Medical Journal; July, 1910; Vol. LIII, No. 7; p. 223.

7. Allbut and Rolleston. System of Medicine. Macmillan & Co.; London, 1910; Vol. VIII, p. 503.

8. Spratling. Epilepsy and Its Treatment. W. B. Saunders Co., Philadelphia, 1904; p. 445.

[Reprinted from The Journal of Nervous and Mental Disease, Vol. 41, No. 5, May, 1914.]

SYPHILITIC TESTS IN LATENT AND TREATED SYPHILIS[1]

By E. P. Corson-White, M.D., and S. D. W. Ludlum, M.D.

One of the most definite characteristics of syphilis is the complete disappearance of symptoms at certain periods, giving rise to a state of apparently perfect health. This condition described by Ehrlich as a "non-sterilizing immunity" may occur spontaneously or as the result of treatment, and it may last for years. For the safety of the patient the true significance of this period is most important. The continued existence of the organism renders the individual always liable to recrudescence of the infection and possibly to the more serious ravages of para-syphilitic diseases.

During the last few years the Wassermann reaction has been used in the diagnosis of many cases, in the demonstration of the origin of many chronic conditions and in the solution of various other problems. The voluminous literature on the subject shows that in the so-called latent periods of syphilis with apparent health only about thirty per cent. of the cases give negative results. Donath (1) has described what he calls a provocative treatment. This consists in the administration of a short course of mercury or a small dose of salvarsan or neosalvarsan. Apparently this procedure has no effect on the nonluetic serum, but changes about nine per cent. of the negatively reacting serums to positives. Donath himself says the influence of the treatment was similar to that of quinine on latent malaria. It has been explained by others in several ways; namely:

(*a*) The liberation of endotoxins by the immediate destruction of a small number of spirochetes gives a sudden intense stimulation to antibody formation.

---

[1] Work done in the Laboratory of Neuropathology of the University of Pennsylvania and in the laboratory of the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases.

(*b*) The dose of spirillicide insufficient to kill the treponema actually stimulates their activity.

(*c*) The endotoxin liberated from the dead organisms gives rise to that substance in the serum on which the Wassermann reaction depends. Most of the other laboratory methods as the demonstration of the spirochete, or the inoculation of animals, etc., fail at this time.

The luetin test devised by Noguchi, besides the decided advantage of a true specificity, gives the most constant results in tertiary latent and late hereditary cases. The technique of this test consists in the intradermal injection of killed cultures of several strains of Treponema pallidum and a control injection of sterile media. In a syphilitic individual this treatment gives rise to a reddish indurated papule or pustule, often surrounded by an inflammatory area. This inflammation increases for four or five days and then gradually recedes. An inflammatory area may sometimes occur around the control injection. The "Umstimmung" of Lesser. This last is probably due to that increased irritability noticeable in the skin of many syphiiltics, while the first, which is always more severe than the control (when the control shows anything at all), is probably a true allergic condition similar to that found in von Pirquet's reaction for tuberculosis.

The importance of a true interpretation of the position of the apparent clinically well individual with syphilitic history, early led many to use the Wassermann reaction to determine whether the facts described in Colle's and Profeta's laws were due to a real immunity or to a latent infection. Opitz (2) was the first to use it for the study of syphilis in women and newborn children. His results stimulated much careful investigation of the field, by means of the complement fixation tests, demonstration of spirochetic and microscopic examination of tissues.

Plaut (3) in his investigation of the rôle of syphilis in the production of feeble-mindedness studied many complete families including those of paretics and tabetics, finding the majority of the members of these families infected. His series unfortunately included only those in which some mental or nervous defect was present in at least one member of the family. In several families with a positive history present in one parent he found that the children clinically positive gave practically always a positive Wassermann reaction, and the reaction was positive in many members of these families that were apparently healthy, but negatively reacting children were also found. These negative results appeared as a rule at one or the other end of the line, although in some cases the negatively reacting child might be between two positives. Knoppelmacher and Lehndorf (4) found that fifty-six per cent. of the mothers of syphilitic children were positive and many more reacted positively if tested within a few months of the babies' birth. Müller and Reich (5) found negative reaction in some children of syphilitic mothers, and Boas and Thomsen (6) in a similar series found that some children which were negative at birth became positive later. Baisch (7) made a very complete study of 140 mothers of children manifestly syphilitic or showing spirochetes in the tissues. He obtained a positive Wassermann reaction in 102 of these women, and demonstrated the Spirochæta pallida in the maternal position of the placenta in twelve of the negatively reacting mothers. He concludes for his study, and it certainly appears from most of the evidence, that the mothers of syphilitic children and the children of syphilitic parents are really infected, fifty-two per cent. of the cases giving positive Wassermann results with the original Wassermann technique, a result which can be slightly increased with the one unit or the Noguchi technique. The true position of the negatively reacting children however is not definitely cleared up by the Wassermann reaction.

It is impossible to accurately state the length of a latent period. A positive reaction may be obtained when the resistance of the body is lowered by some cause or other and the latent individual develops an active syphilis.

In treated cases, having once had a positive reaction, the quantitative reaction is the best index of the intensity of disease and of the effect of the treatment.

Citron (8) was the first to study the effect of treatment, finding a loss of the reaction in sixteen per cent. of the cases. The change varies in different individuals, disappearing quickly in some and persisting even after rigorous treatment in others. Irregular and inadequate treatment never gives rise to negative results. There is a tendency in most cases for the reaction to disappear with continued treatment and to reappear if treatment is discontinued. With salvarsan and neosalvarsan most of the reports are studies of the effect of one injection. The most interesting of these investigations were those of Noguchi and Bronfenbrenner's (9) quantitative study of 102 serums, before and at weekly intervals after the injection of salvarsan, and Craig's (10) analysis of 225 cases according to stage of disease, intensity of reaction and method of treatment. These reports show that the reaction may become negative in from ten to thirty days after the injection, usually in the third week, and that a large percentage of these negatives relapse. In all cases there is a *definite quantitative* reduction in the strength of the reaction, more marked in primary and early secondaries, at which stage relapses are less frequent. Still more favorable are the early cases with weak positive reactions. The reduction in the reaction is roughly comparable to the clinical response but is in every case slower.

The tables which we submit are collected from the work of the last two and a half years and include cases from many sources. They are grouped to show primarily the significance of negative reactions in families of infected individuals (Table I), and in apparently clinically well-treated patients with luetic histories (Table II). Incidentally, to demonstrate the comparative serological value of various methods of treatment (Table III). While many patient were examined, we have included only those in which we were able to make a luetin test and in which we had a record of carefully titrated serum reactions over a period of at least three months. The great majority of these cases were those not yielding quickly to treatment (late secondaries and tertiaries) and the rather high per cent. of positives in the treated cases is due to the fact that the titration was as fine as possible and anything not absolutely negative was counted as positive. From

TABLE I

	No.	Persons	W. R. +	N. R. +	Luetin
Families of syphilitics	17	31	26	27	29
TABETICS:					
sterile	7	7	1	1	5
c. abortion	4	4	1	1	2
children abortion	17	47	21	22	22
children	8	28	9	11	11
PARETICS:					
sterile	4	4	0	0	1
abortion	9	9	4	4	4
children abortion	3	7	4	5	5
children	5	14	2	2	2

these tables it is evident that the negative reactions occur slightly earlier with the Wassermann technique than with the Noguchi and both give earlier negatives than can be demonstrated with the luetin. Excluding the cases of paresis and tabes in which the luetin test results were irregular, we found that every form of treatment reduced quantitatively the antibody content of the serum. The reduction is less marked in cases starting treat-

TABLE II

Cases	W. R.	N. R.	Time	Luetin
42	—	—	3 mo.	1–
27	—	—	6 mo.	1–3±
14	—	—	9 mo.	5–2±
17	—	—	1 yr.	11–2±

TABLE III

Cases	W. R.	Treatment	Stage	W. R. +	N. R. +	Luetin
7	xxxx	Hg. inc. 3 mo.	2dary	7 av. x	7 av. xx	7
11	xxxx	Hg. inc. 3 mo.	3-ary	11 av. xx	11 av. xx	11
18	xxxx	Hg. inc. 3 mo.	tabes	1 sav. xx	1 sav. xx	11
8	xxxx	Hg. inc. 3 mo.	paresis	sav. xxxx	sav. xxxx	6
12	x	Hg. inc. 3 mo.	2dary	5 av. *x*	5 av. *x*	12
9	x	Hg. inc. 3 mo.	3-ary	5 av. *x*	7 av. *x*	9
5	x	Hg. inc. 3 mo.	tabes	3 av. *x*	3 av. *x*	2
3	xx	Hg. inc. 3 mo.	paresis	3 av. xx	3 av. xx	2
8	xxxx	606.4 inj.	2dary	2 av. *x*	3 av. *x*	6
8	xxxx	606.4 inj.	3-ary	2 av. xx	5 av. xx	8
7	xxxx	606.4 inj.	tabes	5 av. xxx	5 av. xxx	5
4	xxxx	606.4 inj.	paresis	4 av. xxx	4 av. xxx	4
9	xx	606.4 inj.	2dary	0	1 *x*	4
9	x	606.4 inj.	3-ary	1 av. *x*	1 x	7
3	x	606.4 inj.	tabes	1 av. *x*	....	1
9	xx	606.4 inj.	paresis	8 av. x	9 av. x	3
3	xxxx	914.4 inj.	2dary	2 av. x	2 av. xx	3
7	xxxx	2	3-ary	3 av. x	3 av. x	6
3	xxxx	2	tabes	3 av. xx	3 av. xx	2
3	xxxx	2	paresis	3 av. xxx	3 av. xxx	2
12	x	2	2dary	0	2 av. *x*	9
8	x	2	3-ary	2 av. *x*	3 av. x	5
2	x	2	tabes	0	1 av. x	2
2	x	2	paresis	1 av. x	1 av. x	0
14	xxxx	914 Hg.	2dary	2 av. x	2 av. x	8
17	xxxx	914 Hg.	3-ary	2 av. *x*	3 av. x	8
8	xxxx	914 Hg.	tabes	5 av. *x*	7 av. x	6
3	xxxx	914 Hg.	paresis	2 av. x	3 av. xx	1
6	xxxx	914–1 mo. hg. 1 mo.	3-ary	2 av. *x*	2 av. x	6
2	xxxx	914–1 mo. hg. 1 mo.	tabes	2 av. xxx	2 av. xxx	2
3	xxxx	914–1 mo. hg. 1 mo.	paresis	3 av. xxx	3 av. xxx	2
7	x	914–1 mo. hg. 1 mo.	3-ary	1 x	4 av. x	4
7	x	914–1 mo. hg. 1 mo.	tabes	3 av. x	3 av. x	7
3	x	914–1 mo. hg. 1 mo.	paresis	2 av. x	2 av. x	1

ment with strongly positive (xxxx) reactions (over two units) of antibody than in those with weak reactions (x), that is one half a unit or less. It is much less in late stages than in early stages. Slow reductions are apt to be the rule in paresis and occur not infrequently in some cases of tabes. Kaplan (11) called attention to this type, calling them the "Wassermann fast tabes." The most marked reduction in the reaction appears in those cases treated with mercury and salvarsan or neosalvarsan, and next in those with salvarsan or neosalvarsan alone. The difference between those two is slight. Salvarsan apparently acts more rapidly than the neosalvarsan, although apparently the neosalvarsan remains longer in the blood than the salvarsan. Mercury gives a slow steady diminution. The serum in patients after long continuation of any type of treatment may show a progressive slowing of the reduction of the antibody content and in some cases even a stationary period.

In conclusion, negative reactions do not necessarily mean absence of syphilitic infection or cure, even although the reaction has been negative for considerable time, as shown in Table II. The luetin test, the specificity of which has up to date never been disproved, is less easily influenced by treatment and is to-day the best criterion of genuine cure or absence of infection in our hands.

BIBLIOGRAPHY

1. Donath, K. Berl. klin. Wchnschr., 1909, No. XLVI, p. 2015.
2. Opitz, E. Med. Klin., 1908, No. IV, p. 1137.
3. Plaut. "The Wassermann sero-diagnosis of Syphilis in its application to Psychiatry," New York, 1911.
4. Knoppelmacher, W., and Lehndorf, H. Wien. med. Wchnschr., 1908, No. LVIII, p. 610; Jahrb. f. Kinder., 1910, No. LXX, p. 156.
5. Müller and Reich. Wien. klin. Wchnschr., 1908, No. XXI, p. 282.
6. Boas and Thomsen. Zeitschr. f. Immunitätsforsch., Bd. X, 1911, p. 337.
7. Baisch, K. Munchen. med. Wchnschr., 1909, No. LVI, p. 1920.
8. Citron, J. Deutsch. med. Wchnschr., 1907, No. XXXIII, p. 1165.
9. Noguchi. "Serum Diagnosis of Syphilis," Philadelphia, 1912.
10. Craig, C. F. Arch of Intern. Med., 1911, No. VIII, p. 395.
11. Kaplan. Jour. Amer. Assn., 1913.

COMPLETE BILATERAL ISOLATED PARALYSIS OF THE SEVENTH NERVE

DEVELOPING FOUR MONTHS AFTER THE PRIMARY INFECTION OF SYPHILIS *

HARVEY M. EWING, M.D.
PHILADELPHIA

When one recalls in what close proximity the sixth, seventh and eighth nerves emerge at the base of the brain, it is hard to conceive of a lesion involving one of those nerves without affecting the others, and it is astonishing to think that any disease process could pick out the seventh nerve on each side, cause paralysis of that nerve and leave intact all the others, even the eighth, which lies in contact with it. Yet this has occurred, and there is at present such a case in the Hospital of the University of Pennsylvania.

Present Illness.—The patient, a white man, aged 32, came to the hospital Feb. 20, 1914, complaining of inability to move the muscles innervated by the seventh nerve on each side. He gave the following history of his present illness: About Jan. 1, 1914, while the patient was eating, he suddenly became aware of a sensation of "stiffness," as he expressed it, in the left side of his face. He continued with his meal, but the sensation of stiffness persisted and in a very short time he found that he could not move the left side of his face. The next morning the stiffness and inability to move the left side of his face were unchanged, and in addition he felt dizzy when he stooped over or moved quickly. The patient could not see clearly at a distance, although he says that he could see objects close at hand and could read for a short time, but his eyes would soon "water" and everything would become blurred. There is a history of some dimness of vision in the right eye which has been present since childhood. Otherwise the patient felt perfectly well. Two days after the onset described above, while out driving, the patient became conscious of the fact that the right side of his face felt stiff and he found that he was then unable to move that side of his face. This condition of inability to move either side of his face persisted, and finally he received some sort of electrical treatment which did not affect the symptoms. After the onset the man worked for one week and then the dizziness on stooping caused him to stop work, and he has done no work since then until one week before admission. He has no pain in his face at present, nor has he had any such pain at any time during the present illness. From the onset he had a full, heavy feeling in his head, which he says felt like "something pressing down," and which lasted for one week. At the time of admission he had a dull, aching pain in the left parietal region. The patient is able to chew his food and to swallow perfectly, and fluids do not come out through his nose. There is a buzzing sensation of some sort in his left ear. He complains of no muscular weakness in any part of his body except in his face, and he says that he walks in the dark without difficulty and never stumbles, nor has he ever noticed any sensation of walking on something soft. There is no complaint of any urinary disturbance nor has he any gastro-intestinal, renal, cardiac or pulmonary symptoms.

---

* From the Neurological Department of the Hospital of the University of Pennsylvania.

Previous Medical History.—About Sept. 12, 1913, he had what he calls a "pimple" on the penis, which was very slow to heal under both local and internal treatment prescribed by a physician. In the latter part of December, 1913, and the first part of January, 1914, he suffered with a sore throat and a sore tongue, but there were apparently no other secondary symptoms. He has had gonorrhea. Seldom does he use any alcoholic beverage.

General Physical Examination on Admission.—The man is large, well built, well nourished and his face attracts immediate attention because of its absolutely expressionless appearance. His lower eyelids are everted and there is marked lacrimation, while his mouth droops, giving him a very dejected appearance. There is no drooling of saliva. He is unable to wrinkle his forehead, and when asked to close his eyes the upper lids move down about half way and stop while the lower lids remain everted. If the patient pulls the upper lids all the way down with his fingers, he is then able to raise them again. There is a total inability to wrinkle either side of his face, he cannot draw up or depress either corner of his mouth, and he cannot smile or whistle or make any movement with his lips. When asked to blow out a match the only motion of his lips is that due to the expulsion of air, and he is unable to extinguish the flame. The lower jaw can be moved up and down and sideways easily, and the tongue is readily protruded in the midline and moved freely in all directions. He had no difficulty in chewing his food. The eyes are moved freely in all directions and there is no nystagmus or exophthalmos. The pupils are equal in size and react readily and equally to light and in accommodation. The sensations of touch and pain are normally acute in the face. The biceps and triceps tendon reflexes are normal in promptness and extent, and sensation to pain and touch is normal in the hands and arms. In the lower extremities the patellar and Achilles tendon reflexes are normal on each side, and there is no ankle-clonus or upward turning of the great toe when the sole of the foot is stroked.

The sensation of pain and of touch is normal in both lower extremities. When the patient stands erect with toes and heels together there is no swaying, and no ataxia is detected by the finger-to-nose test. His gait is normal. Nothing abnormal was found on examination of the heart, lungs and abdomen. The urine examination has shown nothing abnormal.

Blood Examination.—The following condition was found: hemoglobin, 75 per cent.; red blood-corpuscles, 4,600,000; white blood-corpuscles, 12,800. The differential blood count was as follows: polymorphonuclear leukocytes, 84 per cent.; lymphocytes, 14 per cent.; large mononuclears, 0 per cent.; transitionals, 2 per cent.; eosinophils, 0 per cent. Feb. 21, 1914, the Wassermann reaction was tried on a specimen of the patient's blood-serum by Dr. John L. Laird and reported to be strongly positive.

Examination of the Eyes.—Dr. George E. de Schweinitz and Dr. Carl Williams reported the following findings: The pupils were equal and reacted normally to light and in accommodation. Vision in the right eye was slightly blurred for details and normal in the left eye. In the right eye is a faint corneal nebula. The fundus of each eye is healthy. Double orbicularis palsy was noted. Vision in the right eye is 4/150, in the left, 6/6.

Examination of the Ears.—Dr. Butler did not find any involvement of the eighth nerve. The drumheads were normal except for slight retraction of the left one. Before inflation hearing as tested by the tuning-fork on the right side was 9/14 and on the left side 8/14. Inflation, however, brought the hearing on the right side up to 13/14 and on the left side to 11/14. The Galton whistle was heard at 0.7 turn on each side, and ability to hear the C_4 fork was normal on each side. Low tones were heard at 3/4 on each side. Air conduction was found to be better than bone conduction. The Weber test was to the left and no involvement of the vestibular branch of the eighth nerve was detected by the revolving-chair test. Some congestion of the eustachian tubes was found, and this was thought to be sufficient to cause the slight diminution in acuteness of hearing. Dr. Butler concluded that there was no involvement of the eighth nerve on either side.

The Electrical Reactions of the Facial Muscles.—These were tested by Dr. J. W. McConnell, and it was found that the reaction to the galvanic current in the muscles of the right side of the face was in response to 5 milliamperes of current, while on the left side of the face 8 milliamperes of current were required to elicit reaction. On both sides of the face the reaction showed the vermicular, slow quality of degeneration. The serial reactions of galvanic current showed the anode closing contraction to be greater than the cathode closing contraction. There was no response on either side of the face to breaking the current. There was no reaction to stimulation by the galvanic current over the nerve. Taste sensation for sweet and salt is absent on the anterior two-thirds of the tongue.

This case, then, is certainly one of involvement of both seventh nerves below the nuclei and probably after the emergence of the nerves at the base of the brain. If the lesion were in the medulla we would find also some involvement of other tracts, but there is absolutely no evidence of such a condition. Both of the motor tracts have escaped, although almost always in syphilitic disease of both seventh nerves there is also an affection of one or both of these tracts. The sixth nerve, lying as it does quite close to the seventh, has escaped, and there is no evidence whatever to indicate that any other of the cranial nerves is involved. Even the eighth nerve, which is in absolute contact with the seventh at its emergence, has entirely escaped on both sides.

Such a paralysis as we have in this case would be very interesting if it affected the nerve only on one side, but here we have both seventh nerves involved, and they alone of all the other cranial nerves, and furthermore, each is affected in all of its branches.

As a rule, cerebral syphilis is an insidious condition, creeping on slowly; but in this case its onset was very rapid and the time of its appearance early, coming with the secondary symptoms. Then again, the nerve on the right side was involved only two days after the paralysis of the left side, the whole clinical picture being complete in less than three days, and it had neither progressed nor receded up to the time of the patient's coming to the hospital. So this astonishing rapidity of onset and progression after the infection is one of the most remarkable features of the case.

Although such a precipitate onset is most unusual, yet it does occur, and indeed there is in the records of the dispensary for nervous diseases of the Hospital of the University of Pennsylvania and reported by Dr. William G. Spiller, another case showing just this feature. The history is in brief as follows:

The man, a negro, aged 22, on about Jan. 19, 1909, that is, two months before admission, had what he called a "chancre" on the penis. Here, as in the other case, the history of luetic infection is clear. Approximately one month after this "chancre" was acquired and while washing his face, he discovered that he could not close his right eye. On examining his face with the aid of a mirror he was surprised to find that his mouth was twisted to the left side. This paralysis seems therefore to have been quite as sudden in onset and to have followed the initial lesion even more quickly than in my case. Ten days after the paralysis of the right side of his face he became aware that the left side of his face was similarly affected, and when he presented himself at the dispensary he was unable to wrinkle either side of his forehead and could not completely close his eyelids.

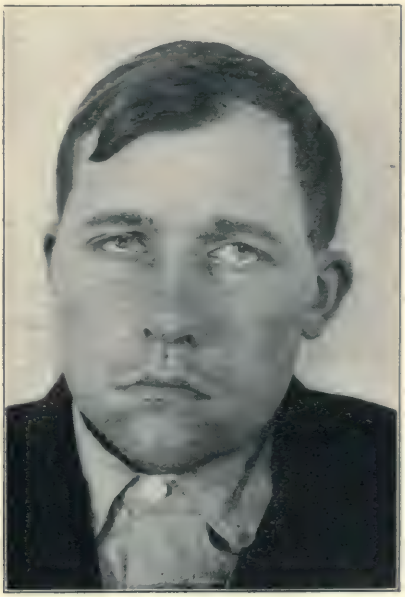


Fig. 1.—Patient at rest.

The involvement of the opposite facial nerve in this case was not so rapid as in my own case, but the paralysis was just as complete on both sides. There was an entire absence of the nasolabial fold on each side, and the man was not able to elevate either corner of his mouth, which hung open at all times. Food collected in both cheeks and also back of the lower lip. There was no loss of sensation for pain and touch in the face, nor any pain. The ocular movements were prompt and free in all directions and the pupils were equal in size and reacted readily and equally to light and in accommodation. Biceps, patellar and Achilles tendon reflexes were all found to be exaggerated on both sides, and a bilateral ankle-clonus was found. There was no upward turning of the great toe on the stroking of the sole of either foot.

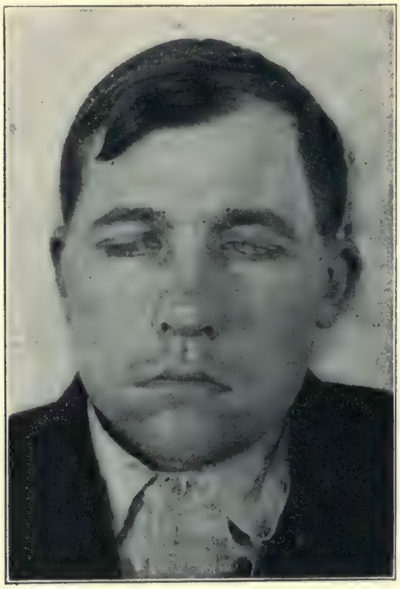


Fig. 2.—Patient attempting to close his eyes and draw up the corners of his mouth before any effects of treatment were noticed.

Tested by the electric current there was found to be no contraction of the muscles on either side of the face to the faradic current when applied over the facial nerve or when applied over the muscles themselves, except a slight contraction of the frontalis muscle. There was no contraction of the facial muscles to stimulation over the nerves by a galvanic current, but a tardy response was obtained when applied over the muscles. Anode closing contraction was nearly equal to cathode closing contraction on both sides of the face.

In Spiller's case there was no history of secondary symptoms of syphilis, but one or two weeks before the onset of the paralysis a stiffness of the neck was noted. Of these two cases the first is the one to which I wish to call particular attention, not only because it presents a very interesting and uncommon clinical picture, but also because of the fact that under treatment there has been some return of power in the muscles affected.

Treatment in Author's Case.—Antisyphilitic treatment was begun Feb. 25, 1914, and since that time the patient has received two intravenous injections of neosalvarsan in doses of 0.45 gm. each. This was supplemented by inunctions of unguentum hydrargyri, 1 dram a day. March 16, 1914, the salicylate of mercury was substituted for the ointment and given by intramuscular injections in doses of 1/10 grain every fourth day. The patient has now had three of these injections. Galvanic electricity is also being used daily over the muscles affected.

At present he is able to close his eyes more completely than when he was admitted, and can make slight upward motion at both corners of his mouth.

[Since the writing of this report the patient has had a further manifestation of secondary syphilis in an extensive loss of hair from his head. He has also had a remarkable return of power in his facial muscles and, at present, can draw up the corners of his mouth to almost a normal degree. He has well-marked nasolabial folds and can wrinkle his forehead very slightly. He has had a fourth injection of 0.45 gm. neosalvarsan.]

In this case I would call attention especially (1) to the clear history of syphilitic infection corroborated by the Wassermann reaction; (2) to the nice selection of each seventh nerve in all its branches, with the escape of all other cranial nerves, and of the motor tracts which are almost always affected in syphilitic paralysis of the seventh nerve, and (3) to the rapidity of onset after the infection and the short interval that elapsed between the paralysis of the seventh nerve on the left and on the right side.

I am indebted to Dr. William G. Spiller for his aid in the study and preparation of this case.

3400 Spruce Street.

Reprinted from The Journal of the American Medical Association
May 9, 1914, Vol. LXII, pp. 1459-1461

Copyright, 1914
American Medical Association, 535 N. Dearborn St., Chicago

Extracted from the American Journal of the Medical Sciences,
January, 1914, No. 1, vol. cxlvii, p. 29.

SOME CAUSES OF DISAPPOINTMENT IN OPERATIONS ON BRAIN TUMOR.[1]

By William G. Spiller, M.D.,
PROFESSOR OF NEUROPATHOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

The discussion on brain tumor introduced by Bruns and Tooth[2] at the recent International Medical Congress, called forth some discouraging statements. Bruns said that about 30 out of 100 brain tumors are so situated that a radical operation may be advised; the localization is accurate and the tumor is accessible. He believed we may expect surgical success in 3 to 4 per cent. of all brain tumors. Tooth studied the records of 500 cases of brain tumor at the National Hospital, obtained during the years 1902 to 1911 inclusive. Of these about half came to operation. He thinks a high mortality is inevitable, but we must not be dismayed by it. We can reduce the mortality by judicious choice of cases for operation and by selection of the operation best suited for individual cases. The immediate dangers common to all growths, all situations, and apparently all operations, are shock, collapse, and respiratory and cardiac failure.

Many causes of disappointment will occur to anyone familiar with operations for brain tumor. The best known are incorrect diagnosis; infiltrating character, large size, and inaccessible situation of the tumor; mistakes in technique and impaired general vitality. Others equally important may be mentioned, but I desire in this communication to speak of the enlargement of the brain caused by tumor, the distortion of the brain especially from tumor in the posterior cranial fossa, and the acute swelling of the brain, probably an important element in the production of hernia cerebri, conditions which usually may be avoided by sufficiently early operation and proper technique.

It is important to remember that when tumor develops in the brain and exists for a long time alteration of the brain structure even in regions remote from the tumor may occur. Failure to take into consideration this alteration may lead to disappointment when operation does not produce the desired result. A tumor may be removed in whole or in part and yet symptoms may persist if a large part of the brain has undergone structural change. Late operation, and most radical operations for brain tumor are late from necessity, is likely at the best to afford recovery with defect, and this defect is dependent in a measure on the altered condition of the brain caused by tumor.

---

[1] Read by invitation at a meeting of the New York Neurological Society with the Section on Neurology and Psychiatry of the Academy of Medicine, November 11, 1913. Lantern slides were used to illustrate the paper, but only a few are represented here.

[2] Proceedings of the XVII International Medical Congress.

Increase of intracranial pressure is believed to be a cause of many of the general symptoms occurring with brain tumor, and it would seem from the writings of certain authors that the increase of pressure is attributed directly to the tumor and varies with the size of the tumor. There is a condition resulting from tumor to which little attention has been paid, and which is as important if not more so than the size of the tumor, viz., enlargement of the brain. This hyperplasia is not directly proportional to the size of the tumor, it may be of moderate degree, with a large tumor or sufficient to cause much enlargement of one cerebral hemisphere where the tumor is small. The hyperplasia may be the result of irritation either from pressure or possibly from some substance elaborated by the tumor, and is caused by overgrowth of neuroglia tissue. The condition is not unknown in medical literature, although little is said concerning it. Oppenheim remarks that the irritation which the tumor causes to the brain may lead to general swelling and increase in the size of the brain, upon which structural change, as proliferation of the neuroglia, follows. The disturbance of the normal relation of cranial capacity to weight of brain leads to functional alteration.

This hyperplasia of the brain may occur with almost any variety of tumor, and is not confined to glioma. It should not be confused with enlargement from glioma tissue, for it has not the appearance microscopically of tumor. It should not be confused with the acute brain swelling of Reichardt.[3]

Tumor external to the brain apparently does not lead to hyperplasia unless it causes much pressure upon the brain. In four cases of tumor of the cerebello-pontine angle I examined in which little pressure upon the brain was produced, no hyperplasia developed, but in another case in which a tumor of this angle caused considerable pressure upon the occipital lobe hyperplasia of the cerebral hemisphere of the same side was pronounced.

According to Reichardt, disease of the posterior parts of the brain in man may cause considerable swelling of the whole brain, and in this way choked disk may be produced. Sudden death may occur from tumor of the pons or medulla oblongata when choked disk has not been present, and this may occur from acute terminal brain swelling.

Reichardt believes the brain swelling as understood by him is not detectable with the microscope. The swelling may be confined to one cerebral hemisphere or to part of a hemisphere. It is probable that the swelling is caused by increased capacity of the brain tissue for fluid. The fluid enters into intimate combination with the brain tissue and is not recognizable as fluid, and the brain may even appear dry.

---

[3] Zeitschrift für die gesamte Neurologie und Psychiatrie, Referate und Ergebnisse, 1911, iii, 1.

Increase in size of the brain may be from various causes: from increase of free fluid in the tissue spaces, from increase of histological elements, from hyperemia, etc., but this is not the brain swelling of Reichardt. The true brain hypertrophy has no relation to the brain swelling, as the latter can appear and disappear rapidly.

The enlargement of a cerebral hemisphere, in which a tumor is situated, by irritation and proliferation of the neuroglia, does not seem to come under the designation of brain swelling as understood by Reichardt.

This hyperplasia of tissue near a tumor is not confined to the animal kingdom, but exists also in the vegetable kingdom. In the illustrations used in a lecture delivered before the Pathological Society of Philadelphia, by Edwin F. Smith, the tissue appeared to me to be proliferated about the tumor strands in plants much as the brain tissue becomes proliferated near a tumor.

The sudden fatal termination that occurs in some cases of tumor probably is a result of this enlargement of the brain, and of interference by the general increase of intracranial pressure with the important functions of the medulla oblongata. In other cases sudden death occurs from hemorrhage or injury of vital parts of the brain by the tumor, the pressing of the medulla oblongata against the edge of the foramen magnum, etc. In my experience, hemorrhage is not the most common cause of sudden death in brain tumor, although I have known it repeatedly to be diagnosed when sudden death occurred.

If the brain be examined by a neuroglia stain there will be found in some instances an overgrowth of neuroglia even in areas at a considerable distance from the tumor. Such increased density of tissue must interfere with the function of nerve fibers, and many of the remote symptoms of tumor probably are from this cause as well as from disturbance of function in associated tracts (diaschisis). The picture given by Rosental[4] in his article on pseudotumor, representing neuroglia proliferation, is much like certain sections from one of my specimens (Fig. 1), but I have not been able to demonstrate proliferation of the neuroglia in all cases. A neuroglia proliferation of moderate intensity is not easily shown even with a neuroglia stain. While I believe that in many cases the permanent enlargement of the brain in association with brain tumor is the result of proliferation of the neuroglia, in whatever way it may be produced, I wish to emphasize the fact that this enlargement of one cerebral hemisphere even if produced in some other way than by proliferation of neuroglia is likely to cause symptoms in addition to those caused by the tumor.

---

[4] Zeitschrift für die gesamte Neurologie und Psychiatrie, 1911, vii, No. 2, 170.

Some of the remote symptoms may be produced by changes in the nerve cells from the tumor. Redlich[5] found in some cases of brain tumor with mental symptoms alteration of the nerve cells and nerve fibers in pieces taken from different parts of the brain, but he could not find the changes in the glia spoken of by Reichardt, and no increase in neuroglia fibers except near the tumor. The changes Redlich found resembled those of senility, and did not satisfactorily explain the mental symptoms associated occasionally with brain tumor.

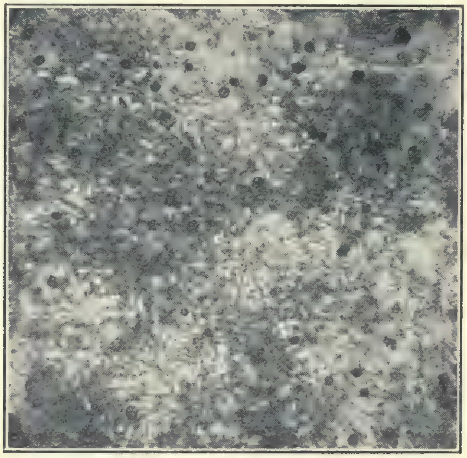


Fig. 1.—Photograph of a section from the centrum semiovale below a small tumor of the parietal lobe. The whole cerebral hemisphere of this side was much enlarged. The neuroglia is shown proliferated in irregular masses. (Mallory's neuroglia stain.)

It may be that hyperplasia of the brain is an important cause of impairment of mentality in some cases of brain tumor. I have known large endotheliomas to exist without any impairment of intellectual power, and in such cases one finds that the tumor has grown slowly, has caused much atrophy of the underlying brain, but may not have led to hyperplasia of a large part of the brain. Some of my cases of enlargement of a cerebral hemisphere presented symptoms which it would be difficult to attribute to the tumor directly. In one, a glioma (449) (Fig. 2) of the pons caused considerable enlargement of one cerebral hemisphere. The patient presented some symptoms which it would be difficult to explain entirely from a pontine lesion. She was hungry all the time and childish. Excessive hunger as a sign of brain lesion I have observed several times.

In another (422) (Fig. 3), a glioma was found in the region posterior to the right optic thalamus. The portion of the right hemisphere in front of the tumor was considerably enlarged. The speech was slurring, and test words were pronounced with difficulty. The memory was poor and the man was easily confused. He acted queerly, attempted to disrobe in public, and did not know how to put on his clothing. He had been alcoholic, so that the effects of alcohol cannot be eliminated, yet I believe that some of his symptoms may have been caused by the hyperplasia.

---

[5] Obersteiner's Arbeiten, vol. xv, part 1.

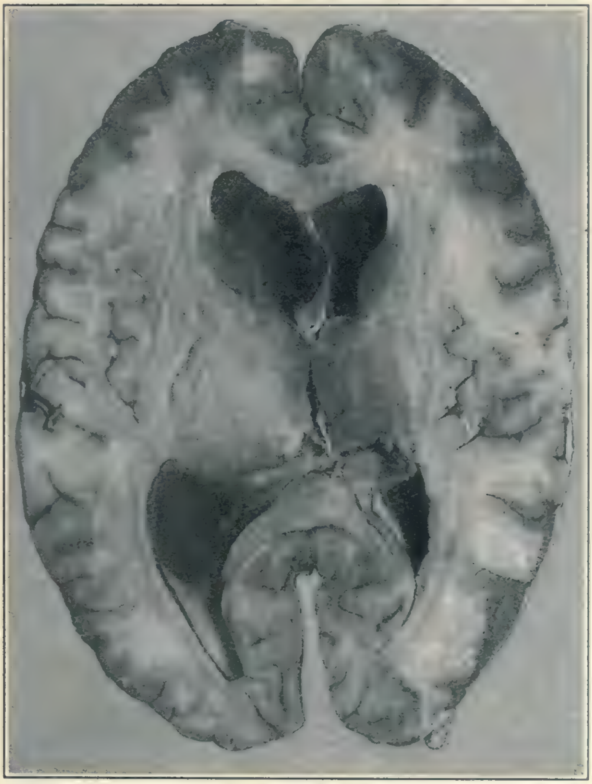


FIG. 2.—Enlargement of one cerebral hemisphere caused by a glioma of the pons.

Another case was interesting in that death occurred very suddenly; the tumor was small, but the whole cerebral hemisphere of the same side was much enlarged. In still another (Keesler), sudden death occurred and a small tumor was found in the left lateral lobe of the cerebellum, and another, also small, was found in the right motor region of the cerebrum, but no hemorrhage was revealed.

It is important to state that the enlargement in all these cases was not from distortion as a result of pressure from a tumor. The parts preserved their relative positions, but were considerably enlarged.

Indeed one of the greatest enlargements of the brain I have observed was from a small tumor of the parietal lobe measuring in a transverse section of the brain 2 x 3 cm. The enlargement of the cerebral hemisphere on the side of the tumor was as great as that represented in Fig. 2. Fig. 1 represents a section from this case.

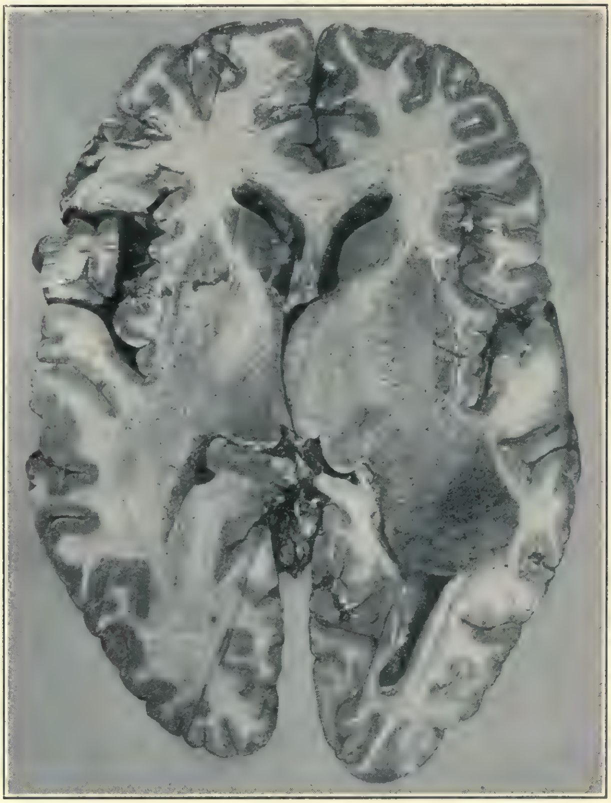


FIG. 3.—Glioma in the region posterior to the right optic thalamus. The same side in front of the tumor is much enlarged.

Distortion of certain parts of the brain is a grave complication of tumor. I have already discussed this in a recent paper in *Brain*. The displacement of the cerebellum and occipital lobes complicates operation, as sudden relief of pressure probably has injurious effects on parts distorted gradually. The medulla oblongata may be so distorted that with the upper cervical cord it forms a half circle. This occurred in a brain from a patient in my service at the Philadelphia General Hospital (Fig. 5). Cranial nerves in the region of the medulla oblongata are liable to be stretched gradually by such distortion, and the effect of this stretching on the pneumogastric may be serious. The distortion in this case was explained by the pressure from a large cyst occupying the greater part of the left cerebral hemisphere, communicating with the lateral ventricle of the same side, and causing atrophy by pressure of the left cerebellar hemisphere. The cyst evidently was from occlusion of the anterior cerebral artery, as its extent corresponded to the distribution of this artery as given by Beevor. One might well consider whether it would not be desirable to delay the removal of a large tumor causing much displacement of the brain, after it had been revealed at operation, until a later operation had given time for the brain to readjust itself in some degree to the changes of pressure following the opening of the dura.

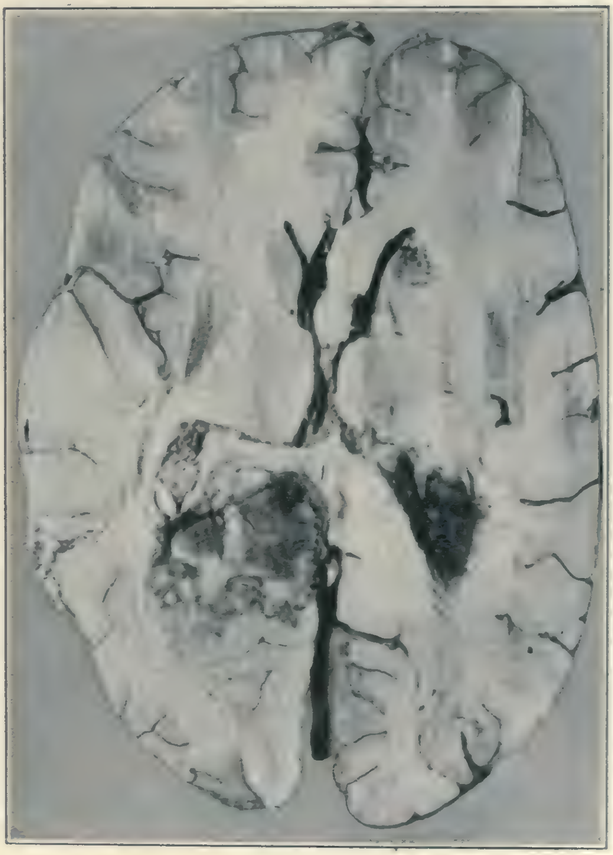


Fig. 4.—Tumor of the occipital lobe. The cerebral hemisphere containing the tumor is much enlarged.

Hernia cerebri is a serious complication of operation for brain tumor. Where it results from decompression it has been supposed to be indicative of the degree of intracranial pressure and to show to what extent intracranial pressure has been relieved. This view is not entirely correct. Intense intracranial pressure possibly may be more liable to cause hernia than intracranial pressure that is more nearly normal, and yet much hernia may develop where there has been no decided increase of intracranial pressure. A large hernia occurred in one of my cases in which a thrombus of the inferior petrosal sinus of long duration had caused atrophy of the adjoining pons, the intracranial pressure could not have been greatly increased, nevertheless a large hernia developed. It seems probable that in such cases acute swelling of the brain in the sense of Reichardt may be responsible for the hernia.

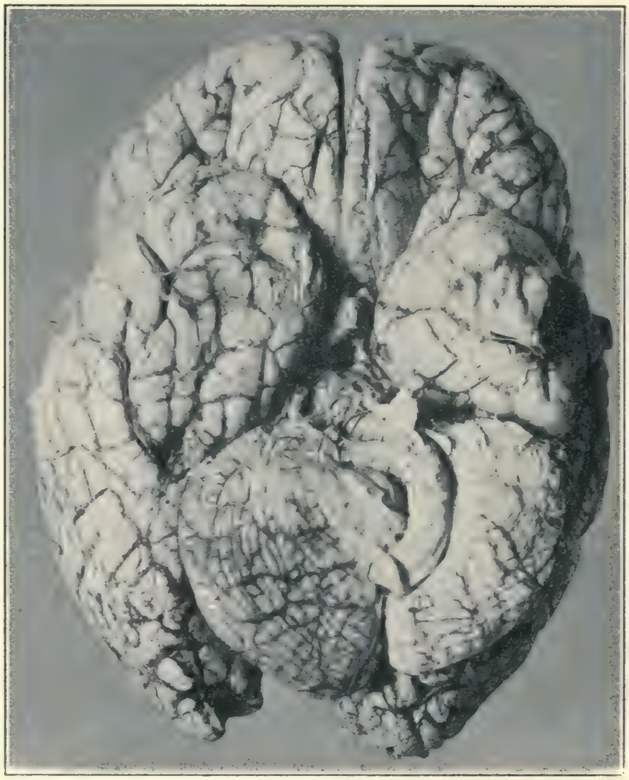


Fig. 5.—Atrophy of the left lobe of the cerebellum and distortion of the medulla oblongata, caused by a large cyst of the left cerebral hemisphere. Displacement of the medulla oblongata of this degree is likely to affect the cranial nerves connected with it.

A hernia may cause pressure against the edges of the opening in the skull, with occlusion of vessels and softening of the parts of the brain implicated in the hernia, causing in this way hemiplegia and other grave symptoms.

I am indebted to Dr. A. J. Smith for the photographs.

A Consideration of Some of the Phases of the Insanity Problem.

BY JOHN H. W. RHEIN, M.D.,
Philadelphia.
Professor of Diseases of the Mind and Nervous System at the Philadelphia Polyclinic and College for Graduates in Medicine; Neurologist to the Howard Hospital; Visiting Physician to the Philadelphia Home for Incurables, etc.

(Presidential address before the Philadelphia Neurological Society, January 24, 1913.)

Reprinted from Pennsylvania Medical Journal, May, 1913.

In selecting a subject for my address to you to-night, I found myself lacking in any laboratory work which was complete enough or sufficiently important to lay before you. I have therefore hit upon a subject which has always deeply interested me, and which has, more to-day than ever, seemed to me to be of the deepest importance, not to say interest, not only to the medical fraternity but equally to the general community. Should I apologize for presenting before a neurological society a paper dealing purely with disease of the mind? I think not, for to me it seems essential that the neurologist should consider, within his specialty, diseases of the mind as well as the functional and organic diseases of the nervous system. I do not see how they can be separated. Moreover, the trained neurologist is best fitted to approach the study of insanity by very reason of his familiarity with laboratory work in which there lies at least some of the hope of the solving of problems arising in relation to the study of insanity. By a trained neurologist I mean one who is a laboratory worker and at the same time a clinician. But so far the laboratory has taken a minor part in the work.

It is to be regretted that the young man in this country who is inclined to scientific research in the laboratory has so little encouragement. Purely scientific results are listened to on the whole with little interest in societies and it is with difficulty that reports of certain types of neuropathological work find a proper medium for publication and when published are too frequently read indifferently.

In the main the neuropathology of organic nervous diseases has been fairly well worked out, and while the training which comes to one occupied in such work is broadening and has a beneficent effect upon the worker, his efforts on the whole rarely produce results especially directed towards the discovery of the causes of, or a cure for, organic diseases of the nervous system. I speak more of the laboratory conditions as they exist generally in this country where experimental work has not yet been extensively done.

It seems to me that the younger men inclined to do laboratory work should be not only encouraged but urged to study the neuropathology of insanity to a greater extent than is being done at the present time. And the work should not stop here, but should be in association with clinical observations as well as biochemical, physiologicochemical and psychological studies. The neurologist needed in this work must be not only a clinical neurologist but an internist. He must be capable of interpreting the findings of the laboratory of physiological chemistry and the laboratory of psychology, and furthermore he should not only be familiar with the sociologic conditions but be able to recognize the economic, legislative and pedagogic aspects of the problem. Only the man who is so fitted can bring with him to the study of diseases of the mind and nervous system an equipment which is to-day a necessity, nay, an imperative demand.

What greater problem faces the neurologist at present than the prevention and cure of insanity and what field of study is broader but, alas, so far, less well worked out?

If insanity is not increasing it is certainly not decreasing to any encouraging extent.

It is a question whether insanity is not increasing in this country. It is extremely difficult to get accurate statistics to show exactly whether this is true or not. The Census Bureau[1] in 1910 canvassed 372 institutions in the United States, and it was found that the number of insane, January 1, 1910, was 187,454. The number of insane cared for in institutions therefore in continental United States in 1910 was greater than the population of the city of Columbus Ohio. There were 37,303 more insane in the United States than in January 1, 1904. There was also an increase of 10,981 in the annual number of commitments. The report of the Census Bureau showed that while the population in the United States increased about eleven per cent. between 1904 and 1910, the population in insane asylums increased about twenty-five per cent. It was the belief of Dr. J. A. Hill, chief statistician, that this increase in the number of commitments was possibly not due to any considerable degree to an increase of insanity but rather to the fact that more insane were cared for in institutions.

The causes of this increase will in part also be found to be due to the earlier recognition of insanity and to the fact that there is less general objection on the part of the public to sending their insane to institutions.

In this connection you will be interested to learn that the study of the tables given by the Board of the Commission of Lunacy in New Hampshire in 1910 shows that the number of insane in that state is increasing in a considerably larger ratio than the population. It is further interesting to note that in Mississippi, twenty years ago, there were less than 200 negroes confined in asylums, while in 1911 there were 700 negroes in one institution alone at the same time that many were confined in jails.

Whether insanity is actually on the increase generally or not, the fact remains that there is an increase in the number of the insane confined in institutions, and it is really this fact with which we have to deal, in large part, as to our responsibility towards the problem of insanity as far as the duties of the state are concerned.

The study of insanity is almost an untrodden field in this country. I say this in spite of the fact that here and there throughout the United States active steps have been taken in the right direction to properly deal with this problem, and to this reference will later be made in some detail. The study of insanity is certainly in its infancy. Wernicke said in 1900 that psychiatry was practically 100 years behind the rest of medicine.

If progress is to be made along the lines of psychiatry, many radical changes must be made in the manner in which the state at present cares for the insane. It is a sad commentary upon our social conditions to be obliged to admit that acute cases of violent insanity which fall to the state's care are often first received in prison cells. There is still a statutory law in Kentucky which does not recognize the insane as sick persons and there are in the United States at least fourteen states who either temporarily or for a long time confine insane patients in jails and almshouses. Furthermore, there is scarcely a state in the Union which has adequate facilities for housing its insane, and there is not one state which is doing all that can be done to help this unfortunate class of patients. It should be noted here that Wisconsin is perhaps the only state which has more than enough hospital accommodations for its insane.

On the whole the state insane hospitals throughout the country are overcrowded, and it goes without saying that this fact alone interferes with the proper care of the patients there confined. Moreover, the number of medical attendants in these institutions is entirely too small. The average medical attendants to patients in state institutions in the United States is about 1 to 200, but there are at least seven institutions in which there is only one medical attendant to 300 to 400 patients. Outside of the psychopathic institutions there is no state institution in the United States where the proportion of medical attendants to patients is less than 1 to 120.

When it is taken into consideration that the medical attendants have a large part of their time consumed in administrative work, one can readily understand how impossible it is that under such conditions any satisfactory progress could be hoped for along the lines of investigations as to the cause or the treatment of mental disease.

Moreover, few of the state hospitals in the United States have proper laboratory facilities and perhaps none outside of the psychopathic institutions which have recently sprung into existence has complete laboratory equipment for the up-to-date study of mental disease. Where laboratories do exist one of the greatest difficulties is to get the proper sort of men to work in them. As a matter of fact, without the laboratory and the proper laboratory man, little advance may be expected. The material is abundant but the facilities for study insufficient.

The inadequacy of the nursing staff is also an obstructing factor. While in some institutions (Maine, Government Asylum for the Insane, New Hampshire and Massachusetts) the attendants are in a ratio of one to eleven, to one to forty, a fair average being about one to fifteen. In few of the institutions moreover is much attention given to the training of the attendants.

The treatment of the insane in state institutions has always been inadequate, and the care of the patients has been largely custodial. Some effort in recent years, however, has been made to employ modern therapeutic measures and the introduction of hydrotherapeutic apparatus is probably the most important step in this direction.

In twenty-nine of the states out of forty in which this question was investigated, one or more hospitals for the insane had installed modern hydrotherapeutic apparatus.

Too much emphasis can not be placed upon the fact that the prognosis of insanity is not as bad as hospital records would indicate and is very often affected in institutions by a lack of therapeutic facilities.

Occupation of the insane in asylums, which is looked upon as of great value in the treatment of mental disease, consists mainly of perfunctory efforts at employment in the workshops of the institutions and working on the farm. In a few of the institutions teachers are employed to instruct the patients in basket weaving, needlework and other manual employment, but in nineteen states of thirty-seven investigated no provision whatever was made for the employment of the patients. As for the care of the criminal insane in this country it may be unhesitatingly asserted that the conditions are far from satisfactory. In spite of the widespread recognition of the needs of separate hospitals for this class of patients, in twenty-eight states the criminal insane are confined in the state hospitals for the insane. In Maine there is a separate building connected with the Maine Insane Hospital for this class of patients and in Vermont and Minnesota they are placed in special wards in insane hospitals. In some of the states the criminal insane are still confined in prisons.

At present there are seven states, Pennsylvania, New York, Indiana, Illinois, Iowa, Massachusetts and Michigan, that have criminal insane asylums, while in Oregon and Wisconsin separate hospitals for this class are in the course of construction.

State care of the insane is the popular method of providing for the insane in this country. Only ten out of thirty-eight of the states have county asylums; viz., Minnesota, Tennessee, Colorado, Indiana (state care declared), Iowa, Maryland, Michigan in one county, Mississippi, New Jersey and Pennsylvania.

Much may be said in favor of both systems, but on the whole the state care would seem to favor a systematic and scientific handling of the situation.

Perhaps the greatest step in the advancement of the care of the insane is in the introduction of the psychopathic hospital. This is a phase of my subject the importance of which I especially wish to emphasize and in which I earnestly hope to awaken an enthusiastic interest among the members of this society. I should like to see a movement set on foot to-night the object of which would be to establish in this state a psychopathic hospital, with modern laboratory equipment, with every facility for the management, care and treatment of early cases of insanity. If we hope to make much progress in the great work of reducing the number of insane in our midst we must begin with the early manifestations of mental disease.

I would favor, nay, I would urge, the appointment of a committee to confer with the State Board of Insanity to agitate this plan and to present the matter before the Legislature at an early date. The State of Pennsylvania can not afford to be lagging behind other states and I know of no more fitting source of such a movement than in the Philadelphia Neurological Society.

The most valuable of this type of hospital are those in Massachusetts opened in June, 1912, in Baltimore and in New York, the last two of which, however, not being state institutions. Allied institutions are, however, under state management in Illinois, Michigan, Rhode Island, and California.

In Massachusetts the psychopathic hospital, opened in April, 1912, is intended for acute, incipient and curable cases. The laboratories are well equipped and facilities for instruction are afforded to students, physicians and advanced workers. There is a free out-patient service and a social service. In this state there are also special well-equipped laboratories in six of thestate hospitals.

The policy of the State Board of Insanity in Massachusetts is to provide in its psychopathic hospital, for the care of acute, incipient and curable insanity as far as the first care, examination and observation are concerned. Also to provide well-equipped laboratories with ample facilities for study, to provide opportunities for instruction to students and to be a center for the education and training of physicians, nurses, investigators and special workers. It also provides for social workers and for an out-patient department.

In this connection it will be interesting to state that the further policy of the board is to provide for a mental sanatorium in which incipient mental patients are received for preventive and curative treatment without commitment. It further provides for the colony care which looks after patients who are mentally unfit to return to life in the community but who are able to receive industrial training and occupation under agreeable conditions. Under this system there should be separate homesteads accommodating twenty-five to one hundred patients each, to be under the management of a good farmer, his wife and assistants. The board also provides for an infirmary for the residual class of patients. The state pathologist supervises the research and clinical work at the various state institutions.

The colony idea has developed extensively while the infirmary has been developed only in existing centers. The sanatorium, as far as I am able to learn, has not been completed. The twelfth annual report of the State Board of Insanity of the Commonwealth of Massachusetts for the year ending November 12, 1910, contains very interesting reading and is very suggestive.

In Illinois there is a psychopathic institute connected with the Kankakee State Hospital. It is used as a center of teaching in psychiatry as well as for research work. Physicians are sent there from the state hospitals, where they act as clinical assistants for a period of three to four months. The laboratory work however, Dr. Singer stated in 1910, had suffered on account of the inadequate appropriations made by the legislature.

Michigan established a state psychopathic hospital at Ann Arbor in 1907. It is the first institution of the kind in the country to care for incipient insanity. This hospital lacks special rooms for treatment and exercises and is in need of proper laboratory equipment.

Rhode Island has a reception hospital for acute cases with a clinical laboratory and special rooms for therapeutics and surgery. In the Stockton State Hospital of California there is a new treatment and receiving building with modern equipment. I am under the impression however that the laboratory facilities are insufficient.

In Wisconsin a recommendation was made to the State Board of Control to establish a laboratory at the University of Wisconsin and to provide for a psychopathic hospital but so far neither has been accomplished.

In Oregon there is in the course of construction a new receiving hospital which will be equipped for carrying out the most modern and approved methods of treatment. No special laboratory provision is made however.

The subject of a psychopathic hospital in Oklahoma has been agitated and a bill introduced to provide for one.

A central laboratory has been urged in Ohio and a laboratory has been planned in Maine.

From this it will be seen that there is an earnest effort in several localities to make provisions for the care of insane in the incipient stages of the disease, and to study disease of the mind in a scientific manner. With this plan fully developed the admission to the general hospitals for the insane will thus be lessened materially, as many cures could be effected in this way.

It will be well for a moment to contrast the work done in this country with that done by the Germans. In Giesen[2] there is a clinic of seventy beds for the treatment of incipient cases. In the Munich clinic there are 120 beds and three to four thousand admissions yearly. There is a staff of fifteen medical men and provisions for a nurse to each five patients. There are eighteen tubs for prolonged baths and five movable baths. The clinic is always open, so that immediate treatment can be obtained under the direction of an experienced alienist. There are ample facilities for scientific investigation along chemical lines, in experimental psychology, of conditions of the blood and cerebrospinal fluid and in anatomy. Moreover, every University in Germany has a psychiatric clinic attached.

Thus far I have called your attention to some interesting features of the state care of the insane, referred to some needs generally, laid particular stress on the special demand for state psychopathic hospitals throughout the country and endeavored to awaken an interest among the members of this society in the enormous importance of this subject. Let me call your attention now to certain other phases of the subject of the modern handling of the insanity problem, and review briefly what is being done in a scientific way to further our knowledge of disease of the mind.

Metabolic studies have engaged the attention of chemists for a number of years, but while an enormous amount of work has been done, not a great deal has been accomplished.

In 1897 Schaeffer[3] reviewed the literature dealing with metabolic changes in the insane but found that none of the studies dealt with metabolism as a whole but considered isolated features.

Barnes[4] in 1912 reviewed the whole subject and concluded that the central nervous system in health or disease exerted no direct influence upon the metabolism of phosphorus. Very little results, he found, were obtained from the study of the urine.

The blood changes too, studied thus far, gave little information. Berkley believed that in dementia præcox there was an increase in the mononuclear elements and a compensatory decrease of the polymorphonuclear elements. Fibrin is said to be increased in paresis. Studies made of the cerebrospinal fluid in regard to lecithin, cholin, cholesterin, phosphoric acid and total nitrogen have not proved of much value.

Barnes believed that lipoidal substances were assuming more importance but admitted that too little was known of the chemical nature of the members of this group of substances to estimate their importance in the realms of the nervous system.

There has been found by investigators some disturbance of lecithin metabolism in paresis. Barnes[5] found in his studies of metabolism in general paresis that there was a tendency also towards a low total nitrogen, that the neutral sulphur in most instances was high and the ethereal sulphur low.

Folin's[6] exhaustive studies of metabolism in sixty-seven cases of insanity including manic-depressive insanity, melancholia, alcoholico-senile dementia, dementia præcox and general paralysis forced him to the conclusion that the results show very little that is tangible concerning mental disorders, except that in general paralysis some metabolic disorders may be associated at one stage or another. He found it impossible to associate any specific metabolic changes with any particular form of insanity. As to the variation in the neutral sulphur, as shown by the work of Koch, Pighini and others, Barnes concluded that its full meaning was yet to be discovered.

R. T. MacKenzie Wallis,[7] having studied metabolic change in eighty-six cases, found the excretion of creatinin in the insane generally subnormal and the creatinin coefficient correspondingly low. The indigo in the urine he believed was derived from sources outside of intestinal putrefaction. The neutral sulphur excretion was low and indicates a diminished cellular activity.

Gilikin[8] found a lowering of the lecithin in the bone marrow in three cases of dementia paralytica and complete absence in six cases.

Pighini[9] found in his studies of cholesterin that it was present only in cases of progressive paralysis, epilepsy and dementia præcox, having failed to find it in other forms of insanity.

Shultz and Knauer,[10] as a result of their studies of the carbohydrate metabolism in insanity, concluded that the glycosuria was the result of the psychosis and not the cause.

In six cases in which active degeneration of the nervous system had taken place Coriot[11] found cholin in the cerebrospinal fluid. This substance is one of the katabolic derivatives of lecithin which forms the principal substance of the myelin sheath. The underlying factors which cause lecithin to split up into glycerophosphoric acid, stearic acid, and cholin afford, he suggests, a fruitful study.

Koch's[12] studies in five cases of dementia præcox showed a variation in the neutral sulphur fraction; in other words, a difference in chemical composition of a nature not seen in health or in other mental diseases, and he concluded that there was a metabolic insufficiency in dementia præcox, the exact interpretation of which is at present impossible. The foundation for the deficiency he believes lies in a disturbance occurring in early or embryonic development, by which the brain lacks full metabolic and recuperative power and is therefore handicapped when meeting the strain of adolescent life, deteriorating then and giving rise to the chemical changes described.

So far then, after rehearsing the evidence, who shall say that metabolic studies have helped greatly? At the same time such studies should go on, but in connection with clinical, pathological and psychological investigations.

What have histological studies done towards solving the mysteries of mental disease?

Outside of general paralysis and the changes in the senile brain described by Redlich,[13] in 1889, and later by Alzheimer,[14] Mijake,[15] Fischer,[16] and Acúcarro[17] especially, histologic changes can be said to show characteristic appearances in syphilitic mental psychoses and the idiots' brains only.

No specific histological change has yet been found for all forms of clinical insanity. As Ladame[18] has aptly said, no histological lesion described hitherto is capable of explaining psychic symptoms such as delirious ideas and hallucinations.

Brodmann's "cytoarchitektonik" may give valuable data in the studies of the layers of cells in the cortex, a line of work which has also been followed by Cajal and Campbell.

Psychology is assuming increasing importance in the study of insanity. It is beginning to be more and more recognized that the studies of the normal mental processes should be applied to investigations of morbid mental states, since Kraepelin first called attention to the importance of the study of the principles of psychology as applied to insanity.

Farrar[20] aptly said that the alive brain must be studied, for the dead brain can not show evidences of morbid thought which existed during life.

Meyer[21] in this country has especially laid stress upon the importance of the psychogenic causes of insanity. The psychogenic disorders, he believes, are dependent on conditions which he states can be described in terms of psychobiology.

While psychologists are employed in Germany extensively in the study of insanity, in this country their importance has been neglected. Except in Vermont and in the Government Hospital for the insane, no state in the Union has, in its institutions for the insane, a psychologist attached.

Virginia employs one at times and Illinois has one connected with its institutions for the feeble-minded.

The prevention of insanity seems to me to offer more, in our present knowledge of the subject, towards the relief of the situation than any other feature of his problem. Is there any work of greater importance to the neurologist than this? Are not neurologists generally more interested along lines of work and study relating to organic nervous disease, which offer so little immediate results from an humanitarian point of view?

The prevention of insanity is to be achieved in several ways. The people must be educated to recognize the importance of prevention. The methods by which this may be accompished must be exploited. The vast saving to the state which accrues from measures to prevent insanity must be emphasized. The enormous advantages resulting from the treatment of incipient cases from an economic as well as an humanitarian standpoint must be recognized.

The cost of maintaining the insane in the United States in state institutions is about $50,000,000 yearly. Much of this money could be saved by an active crusade which has for its object the prevention of insanity.

The etiological significance of alcohol and syphilis should be spread generally among the people. Apropos of this you will be interested to hear that Felix Rose of Paris, in a personal letter, made the statement that ninety per cent. of the cases of insanity in French asylums was due to alcohol.

The study of educational measures in regard to children; the early recognition of danger symptoms in growing children under educational pressure; the exploiting of the evils of procreation among the feeble-minded; the early recognition of the neuropathic character; avoidance of head injuries; the value of studies in eugenics and of labor problems connected with children and women; the importance of all these must be brought before the public eye in a vivid and impressive manner.

The public must be reëducated not only as to the importance of these but also to the necessity of having psychiatric hospitals and psychiatric wards in general hospitals where those afflicted with mental disorders may be readily received for treatment in the incipient stages of the disease.

Coöperation in this work with the general practitioner is essential. Furthermore, public lectures should be given and properly censored publications in newspapers should be encouraged.

The state should be urged to give greater support to the scientific aspect of the problem along the lines of prophylaxis as well as treatment. Movements should be inaugurated by every state, such as that made by the National Committee of Mental Hygiene, the Connecticut Society for Mental Hygiene, the Illinois Society for Hygiene and in New York, the State Charities Aid Association.

The chief objects of the National Committee for Mental Hygiene are: "To work for the protection of the mental health of the public; to help raise the standard of care for those threatened with mental disorder or actually ill; to promote the study of mental disorders in all their forms and relations and to disseminate knowledge concerning their causes, treatment and prevention; to obtain from every source, reliable data regarding conditions and methods of dealing with mental disorders; to enlist the aid of the federal government so far as may seem desirable; to coördinate existing agencies and help organize in each state in the Union an allied, but independent society for mental hygiene, similar to the existing Connecticut Society for Mental Hygiene."

The objects of the Connecticut Society for Mental Hygiene, for the establishing of which Beers is responsible, are: "To advise and assist those in danger of nervous or mental disorders, or actually ill; to coöperate with the hospitals in the work of caring for the insane, and to assist the many who are discharged as cured or improved, so that relapses due to avoidable and remediable causes may be prevented; to enlighten the public regarding all phases of mental disorder and the care of the insane, so that it may the sooner abandon the semi-superstitious, if not ignorant, belief that mental disease is a disgrace, and that commitment to a hospital for the insane stigmatizes the patient and his relatives; in other words, to impress upon the public that the insane are sick people, not criminals, and that they must be given the benefits of prompt and intelligent treatment if the many curable cases are not to be deprived of their rightful chance of recovery."

The importance of better and fuller instruction in our medical colleges of this country must be emphasized. In Germany, courses in psychiatry are compulsory but in this country generally little heed is given to the subject in the curriculum. Also instruction should be given in psychology as related to mental disease.

Mental hygiene should become an important feature of the work of departments of public health.

Social service in connection with problems of insanity holds an important place. The care of patients after they are discharged from the hospital and sent home well, which is of such enormous importance, can be carried out through this agency.

A movement to provide after-care for the insane was instituted as early as 1841 by Dr. Falret in France. In this country the importance of this subject was suggested in 1894 by a committee of the American Neurological Association consisting of Drs. Stenton, Dana and Dercum. Associations of this character are in operation in Switzerland and England. In 1902 in Japan a Tokio Ladies' Aid Society for the insane was organized with similar functions. In 1905, Miss Louise L. Schuyler instituted the association in New York which provides after-care for the insane in that state. The object was to form a committee in connection with each state hospital to give aid, advice, assistance, etc., to those discharged from hospitals, and to surround them with an environment which shall give them the best chances. This plan not only helps the individual but anticipates a relapse and thus prevents such a person becoming a public charge again.

The social service also gives aid to patients who have recovered but who are without friends. Furthermore, through this agency home conditions can be studied, histories completed and data relating to heredity and causation collected. It further assists in the reëducation of the family and the community and helps to awaken the interest of the public.

In conclusion, I wish to call attention to another factor which enters into the economic aspect as well as that phase of the subject which pertains to the prevention of insanity in our midst, namely, the alien insane in this country. There has been in the last three years a yearly average of 330 insane, epileptics, feeble-minded, imbeciles and idiots who have been excluded from our ports.[22] In spite of efforts to prevent the entrance of this class of immigrants it has been found necessary to deport within three years of entry about twice this number each year for three years of aliens suffering from epilepsy or some mental affection.

There were in 1903 in this country 47,018 foreign-born white insane and during 1904 there were admitted 13,405 foreign-born white insane in hospitals of continental United States. It will thus be seen that a large proportion of the insane in state institutions is made up of foreign-born whites. The cause of this large number of foreign insane in our institutions needs careful study and investigation. It would be idle to speculate upon the meaning of these figures without an exhaustive study of the conditions surrounding this aspect of the situation but certainly it would appear that something might be done to lessen this number by proper legislation and a more careful study of the mental condition of immigrants.

BIBLIOGRAPHY.

1. Insane in Institutions. Department of Commerce and Labor and Bureau of the Census, Washington, 1911. Preliminary summary for 1910, issued by the Census Bureau.

2. Rows: *Jour. of Mental Science,* 1912, p. 25.

3. Schaeffer: *Monatsh. f. Psych.,* 1897. Bd. II., p. 157.

4. Barnes: *Jour. of Insanity,* 1912, p. 431.

5. Barnes: *Am. Jour. of Insanity,* 1909 and 1910, p. 301.

6. Folin: *Am. Jour. of Insanity,* Vol. 60, p. 699, Vol. 61, p. 299.

7. R. T. MacKenzie Wallis: *Jour. of Mental Science,* 1911, Vol. 57, p. 327.

8. Gilikin: *Biochem. Ztsch.,* 1909, Vol. 19, p. 270.

9. Pighini: *Ztsch. f. Physiol. Chem.* 1909, Vol. LXI., p. 508.

10. Schultz and Knauer: *Allg. Ztschr. f. Psychiat.,* 1909, LXVI., p. 759.

11. Coriot: *Am. Jour. of Insanity,* 1903-4, Vol. 60, p. 733.

12. Koch: *Jour. of Experimental Med.,* 1911, Vol. 13, p. 301.

13. Redlich: In Barrett, *Am. Jour. of Insanity,* 1910-11. Vol. 67, p. 503.

14. Alzheimer: In Barrett, *Am. Jour. of Insanity,* 1910-11. Vol. 67, p. 503.

15. Mijake. In Barrett, *Am. Jour. of Insanity,* 1910-11, Vol. 67, p. 503.

16. Fischer: *Ztsch. f. d. ges. neurol. u. psych.,* 1910, Vol. III., p. 371.

17. Achucarro: Government Hospital Insane, Bulletin No. 2, 1910, p. 81.

18. Ladame: n. Iconag. d. l. Salpetreiere. Maladie du Syst. nerveuz, 1910, Vol. 38, p. 193.

19. Brodmann: In Achucarro, Gov. Hos. Insane, Bulletin No. 1, p. 43.

20. Farrar: *Maryland Med. Jour.,* 1908, Vol. LI., p. 374.

21. Meyer: Psychol. Clinic Phil., 1908, Vol. II., p. 89.

22. The Insane and Feeble Minded, 1904. Department of Commerce and Labor and Bureau of the Census, Washington, 1906.

Reprinted from West Va. Medical Journal, June Issue, 1913

191

The Treatment of the Insane in Psychopathic Wards.

By John H. W. Rhein, M.D.

Professor of Diseases of the Mind and Nervous System at the Philadelphia Polyclinic and College for Graduates in Medicine, Neurologist to the Howard Hospital, Physician to the Philadelphia Home for Incurables, Philadelphia.

Read at the Conference and Exhibit on Mental Hygiene, March 17, 1913.

To understand the real significance, importance and value of the psychiatric ward one must take into consideration to some extent the evolution of the treatment of the insane and must recognize that on one end of a chain is the treatment of the insane as if they had descended to the levels of brutes and animals, that they were witches or that they were abandoned by God and deserved less consideration than the criminal, and at the other end of the chain is the psychiatric ward the most advanced and humane method of dealing with those mentally sick, recognizing that they are patients in the strict sense of the word, sick human beings, and therefore to be treated with the same care and consideration as one suffering from typhoid fever, pneumonia or measles and with no more stigma attached than one afflicted with these diseases suffers when going to a general hospital.

The links in the chain reflect little credit upon either the intelligence or the humanity of society.

To understand what the psychiatric ward really means to the mentally sick let me briefly review the steps in the evolution of psychiatry from antiquity to the present.

In 460 B. C. Hippocrates removed the insane from the care of priests who until that time treated them by ocular consultations in temples dedicated to Esculapius.

The great genius Hippocrates recognized that mental diseases were the result of abnormalities of the brain, and were due to acute and chronic diseases of the organs of the body, the treatment being by medicine, diet and rest.

Aretaeus (60 A. D.), Galen (160 A. D.) and later Aurelianus, a contemporary of Trajan and Hadrian, placed the study of psychiatry on a rational basis, and the latter at this early period advised and practiced the removal of restraint in the treatment of the insane.

In the middle ages medicine and especially psychiatry declined, and insane patients became the victims of superstition and fanaticism and were treated by the priests to whom their treatment was again relegated, as devils and witches and were put to death at the stake or confined in jails.

It is much to the discredit of the human race that these poor unfortunates were victims of witch-trial as long as the latter persisted in the eighteenth century.

With the Reformation, only incipient advances were made in psychiatry, but the lot of the insane remained deplorable until the middle of the eighteenth century, and though physicians recognized that insanity was an expression of a diseased nervous system, that it was curable was yet unrecognized and the jails still were considered the proper place to lodge an insane person.

The cure of the insane was first attempted in England about the middle of the eighteenth century, at St. Luke's Hospital, London.

In France in 1780 Howard and the Emperor Joseph, who were studying the conditions of hospitals and prisons, inaugurated important reforms.

Asylums, however, had been established in Italy in 1352, in Rome in 1548, in Valencia, Spain, in 1409 and in France in 1660, when the Hotel Dieux was used for the care of this class of unfortunates.

It remained for Pinel to really inaugurate reforms, which proved to be permanent, when in 1792 he removed the chains from the insane and treated them as human beings.

However, as late as 1818 Esquirol, a noted French alienist, stated that the insane in France were treated with less consideration than criminals and animals.

In 1845 Griesinger wrote a book which was epoch-making in its beneficial influences and placed the study of insanity for the first time on a scientific basis. (See von Krafft-Ebing Textbook of Insanity, 1904.)

It is only comparatively recently really that insanity has been studied to any satisfying degree in a scientific manner, and it is only in the last few years that the disturbances in the function and the organs of the body in their relation to insanity have been studied in laboratories as well as from the standpoint of the clinician and that the importance of studies from a psychological standpoint has been recognized.

Germany has comparatively recently handled the situation in a thoroughly satisfying manner.

At Giesen, for example, there is an institution for the care of the incipient insane with 70 beds, and in Munich there is an institute where in one year 3,000 to 4,000 cases were treated and with such good results that the building of asylums has been postponed. These clinics are open day and night, so that patients may be received without delay under the care of physicians who are expert in the treatment of insanity. There is this great advantage that no legal document is required.

This brings us to the consideration of the subject of my address, namely, the psychopathic department or ward.

By this I mean a department attached to a general hospital where those afflicted with insanity in its infancy may receive at once the very best, most approved, up-to-date care and attention by skilled physicians, trained to this kind of work. The incipient insane must be looked upon as sick individuals and are to be treated with the same care and study as those suffering from any other disease requiring hospital care.

Laboratories are a most important feature of such a unit. Studies of the blood, cerebro-spinal fluids and the various secretions by bacteriologists, pathologists and chemical physiologists can thus be made. A psychologist should be connected with the department to study the individual in a properly equipped laboratory. This department must be provided with hydrotherapeutic apparatus, occupation rooms, rooms for diversion, and should afford opportunities for outdoor exercise and amusements. There should be sufficient and competent medical attendance and the number of nurses should be adequate. These should be specially trained and knowledge of massage and gymnastics or calisthenic work should comprise a part of their equipment.

Let me call your attention to some of the present features of asylums today in contrast to this up-to-date and much desired method of treating the insane. It is a sad commentary upon our social condition that the first care of acute cases of violent insanity is often received in prison cells.

In 14 states the insane are temporarily or for a long time confined in jails or almshouses.

Wisconsin is the only state that has sufficient accommodation for its insane. Asylums in most of the states are over-crowded.

Furthermore, there is an average of one medical attendant to 200 patients and in some, one to 400. These medical men have their time largely occupied in administrative work and hence can give little time to the study of the cases or sufficiently close attention to their treatment.

Institutions throughout the country lack laboratory facilities, an extremely important factor in the treatment of these cases.

In four institutions in the United States the number of nurses is one to nine patients, but usually the ratio is one to 15 or one to 40.

In 29 of the 40 states investigated only was there installed hydrotherapeutic apparatus.

In only 19 of 37 states was any attempt at occupation made and in none of the remaining states was there any occupation afforded the patients whatever.

These facts are laid before you to emphasize the great need of proper facilities for the treatment of the incipient insane.

As long as the people will allow their insane to remain in jails and poorhouses there they will remain. Not to know of the conditions can no longer be offered as an excuse, for this movement now on foot is intended to show the people the actual state of affairs. But it is really up to the people to see that the legislature provides better accommodation and care for this sad class.

Psychopathic wards in general hospitals are found only in the Albany Hospital and Bellevue Hospital in New York and the St. Francis Hospital, Pittsburg.

In the Albany Hospital there are 32 beds, 3 doctors and 10 to 12 nurses.

In the St. Francis Hospital, Pittsburgh, there is a psyciatric ward accommodating 125 patients, of which, however, 75% are chronic cases. The State of Pennsylvania has appropriated money for building an addition to this, but it is not yet in operation.

There is also a psychopathic ward connected with the Bellevue Hospital of New York.

In addition to these there is at Boston a State Psychopathic Institute, a Psychopathic Institute in New York, and in Baltimore there is the Phipps Psychopathic Institute for the accommodation of 60 public cases and 20 private cases.

In Michigan and Illinois there are somewhat similar institutons, and here and there, in Rhode Island, California, Oregon, Wisconsin, Oklahoma, Ohio and Maine, efforts are being made in this direction.

In conclusion the benefits to be derived from a psychopathic department or institution are (1) first care under ideal conditions. In this way many cases can be cured that otherwise would go to an asylum to remain insane. The percentage of cures would be instead of 20%, as it is in asylums, more than 50%. (2) The avoidance of the stigma which is attached to any one who becomes an inmate of an asylum. (3) The opportunities to study the causes of insanity by the aid of laboratory facilities. (4) The advantage as a teaching centre for physicians, general practitioners as well as for those physicians intending to take up institution work. (5) To act as a centre for distributing information to the public along the lines of prevention of insanity and care of those who have recovered. (6) To act as a centre for after-care societies. (7) In an out-patient department borderline cases may apply for treatment at an early period in their psychosis.

1732 Pine Street.

The Present Status of the Insanity Problem.

BY JOHN H. W. RHEIN, M.D.,
Professor of Diseases of the Mind and Nervous System at the Philadelphia Polyclinic
and College for Graduates in Medicine, Neurologist to the Howard Hospital,
Physician to the Philadelphia Home for Incurables, Philadelphia.

(Read in the Section on Medicine, Medical Society of the State of Pennsylvania,
Philadelphia Session, September 25, 1913.)

Reprinted from Pennsylvania Medical Journal, June, 1914.

It is not the purpose of this paper to discuss in detail the inadequate features of insane asylums especially. It is sufficient to state what is common information that asylums on the whole are overcrowded and their population is increasing out of proportion to the increase of the general population; that they have too few medical attendants; that they are insufficiently supplied with nurses; that they are on the whole lacking in proper laboratory facilities and proper laboratory workers; that only a little over half of the state asylums in this country are supplied with hydrotherapeutic apparatus; that definite employment or occupation of the insane is practiced in only about half of the asylums; that in twenty-eight states the criminal insane are still confined in state hospitals for the insane instead of in criminal insane asylums; that insane are still confined in jails and almshouses in fourteen states of the Union; and, finally, that there is a lack of adequate teaching of psychiatry in medical schools. These are the main weak points in the state's management of the insanity problem to-day and these alone show without further harrowing details of asylum doings, that the provisions for the insane as to care at least are insufficient.

The hope of the future of the insane is inseparably united with definite plans to establish psychopathic departments connected with teaching institutions, preferably a postgraduate medical college, and psychopathic wards in general hospitals. The advantages of a psychopathic department in a teaching institution are that (1) the patients may be immediately placed under proper treatment without the delay incident upon commitment; (2) the stigma of a residence in insane asylums is thus avoided; (3) it affords an opportunity of studying the cases in a definite manner by reason of laboratory facilities and the opportunities of consultations with other specialists connected with the medical school and hospital; (4) such a department may act as a teaching center for general practitioners as well as to afford opportunity of training those physicians who intend to take up institutional work; (5) in connection with such a department an outpatient department is provided, where in borderline and incipient cases the patients may apply for treatment, and finally, the existence of after-care and social service departments.

There is a need of a propaganda of education in order, first, that medical men should acquaint themselves with the facts of the existing conditions and that they may see the need of learning to recognize the early symptoms of insanity and to know what to do at once, when the symptoms present themselves. This educational propaganda should also have for its object the education of the public as to the causes of insanity, the importance of seeking early care for the insane, the need of special facilities for their care and to awaken an interest in the care of the insane after their discharge from the hospital. It is necessary to recognize the need of social workers for the purpose not only of after-care which means protecting the patient from future attacks if possible, but for the purpose of gathering data about home and other conditions surrounding the patient, together with the history of the patient before coming under observation, which could thus be carefully studied for the purpose of accumulating facts relating to causation as well as their bearing upon the management of the case.

The economic aspect of the problem as seen in the saving to the community incident upon prevention, not to speak of the humanitarian viewpoint, is of great importance.

In this connection it is well to mention here the value of movements such as the National Committee on Mental Hygiene, of which there are branches in Connecticut, Illinois, New York and Pennsylvania. In this city last month there was a conference and exhibit on mental hygiene conducted by the Civic Club of Philadelphia, the Public Charities Association of Pennsylvania, the Department of Public Health and Charities and other coöperating organizations, which lasted a week and created an enormous amount of interest and was effectual in the lines in which it was intended to extend its influence.

Lectures were given on prevention, care and the cure of insanity together with discussions of the social evil, eugenics and indeed every phase that the problems present. Thus the public were instructed in these matters and together with the exhibits were given an idea of the possibilities in handling the situation of the insane in our midst.

An extension of these movements to other states is the intention of the National Committee on Mental Hygiene and this is perhaps one of the most far-reaching steps that has been taken to solve the problem.

The importance of early recognition of insanity is of profound significance. Nearly a third of the insane do not enter asylums and are therefore without treatment until the symptoms have existed for almost a year.

The significance of this statement is seen when one reflects that it is estimated that fifty per cent. of the patients coming under early and proper care recover and that the hope of averting terminal chronic mental states in many of the cases of insanity is in early recognition and treatment.

It should be a matter of common knowledge that forty per cent. of the cases of insanity appear to be connected with preventable causes, namely alcohol and syphilis. The solution of the prevention of insanity from this standpoint brings up discussions of broad social conditions which have been already brought out at this meeting in some detail.

The influence of heredity in relation to the feeble-minded is an important aspect of the problems which has also been discussed at length, together with the means of prevention. The state provisions unfortunately are very inadequate, not only in not providing proper housing for this class but in not appropriating a sufficient amount of money properly to carry on the study of the features of the problem pertaining to proper methods of prevention. Indeed, while for the purpose of the education of the public and measures for prevention the state gives but little, millions are spent yearly to house and care for the insane, a large proportion of whom could have been cured outside of asylums.

I want to emphasize the hopefulness of the subject. It should be generally known that what at one time was looked upon as an unspeakable horror, namely to become insane, is no longer such, that many of the insane can be cured and that a large percentage of cases of insanity can be prevented.

Prevention saves money for the community as well as sparing an enormous amount of human suffering and unhappiness.

The state should be as active along those lines as it is in the prevention of the spread of infectious diseases. In fact in the legislature we have the keynote of the situation. The interest of our legislators must be awakened by the community first, however, and, in efforts similar to the conference and exhibit recently held in Philadelphia, we may hope to in part at least achieve this end.

Preventive measures further include a study of the educational plans of children, a study of the response that children make to their surroundings, a supervision by trained medical men of the lives of children who exhibit early morbid nervous manifestations especially if they have inherited, from neurotic ancestry, unstable nervous systems.

Conditions under which children and women work touch the subject of prevention and should be controlled by proper laws.

Eugenics have an important bearing upon the subject of prevention and laws regulating marriages should be favored to the end that fewer unfit shall marry.

A word only as to the astounding fact that in 1903 there were about 50,000 foreign-born white insane in insane asylums. Only 330 were yearly excluded for the last three years on account of mental conditions. The inference is perfectly clear that stricter examination should be made of the mental states of immigrants.

The scientific study of insanity in laboratories has been going on for a number of years, but thus far the subject is still in its infancy in spite of the fact that studies of metabolic changes by physiological chemists and histological changes by neuropathologists have been carefully made in recent years.

A reference at least should be made to the studies of Freud, Breuer, and Jung. Freud's psychoanalysis claims in borderline cases to cure patients suffering from phobias, obsessions and other morbid manifestations, and more recently psychoanalysis has been introduced into the study of actual insanity.

What values are to be placed upon Freud's studies can not at this time be definitely stated. It may be said that the ideas he advanced have not been accepted by many neurologists in this country and abroad.

Somewhat briefly Freud's psychoanalysis consists of awakening certain memories of events occurring usually in childhood of a sexual nature which as time passes have been forgotten. These memories in the nature of a psychic trauma come in conflict with the consciousness which in turn endeavors to prevent these memories from becoming conscious. To become conscious they would produce feelings of shame, reproach or mental pain and it is thus desirable that they do not become conscious.

This constitutes the idea of repression. This combat results in the development of a new complex by means of the process of conversion and the new complex expresses itself in some symptom in the nature of an obsession or other morbid manifestation which is connected with the original painful memory, although it is not ever so recognized by the patient. The same principle has been applied by Jung to explain dementia precox. Freud also applies the same principle in explaining certain forms of hallucinatory confusion. Dreams are also interpreted, Freud claiming that dreams have definite meanings and are the expression of a wish fulfillment. Repression explains the dream as well as the symptom.

The patient undergoing what is termed by Freud the cathartic treatment is placed in a recumbent position and is then questioned daily, for weeks, months and even years in order to recall the suppressed memory. When the repressed memory becomes conscious the symptoms which it caused disappear, producing an emotional feeling during the period that the original trauma was rehearsed but finally disappearing. This process is termed the abreaction.

In conclusion I wish to say that the crusade against insanity is just begun, and the education of the public is gradually progressing but it would progress more rapidly if every general practitioner should interest himself in the problem along the lines suggested in this brief contribution.

Reprinted from the New York Medical Journal for January 31, 1914.

NEUROLOGY AT THE RECENT (SEVENTEENTH) INTERNATIONAL CONGRESS OF MEDICINE.*

Impressions with Comments on Some of the More Important Subjects Presented.

By Charles K. Mills, M. D.,
Philadelphia,
Professor of Neurology, University of Pennsylvania.

PERSONNEL AND GENERAL IMPRESSIONS.

Of the many medical gatherings which I have attended the two most instructive and inspiring were the first meeting of the American Congress of Physicians and Surgeons, held in Washington during September, 1888, and the recent (seventeenth) International Congress of Medicine in London, August 6 to 12, 1913. The first had its origin in the progressiveness which was then beginning to stir the profession in all its departments. The second brought together from all parts of the civilized world the most distinguished representatives of medicine, in whose work the inspirations of twenty-five years earlier had found realization. At the Washington congress cerebral localization received prominent consideration, and in the London gathering cerebellar localization was one of the chief topics.

It was a pleasure to those who had had the opportunity of attending both gatherings to see Sir David Ferrier, who had made important contribution to the congress of 1888, presiding with dignity at the meeting of the section in neuropathology in London. Alert and active, always at his post, it

---

*Read at a meeting of the Philadelphia Neurological Society, November 28, 1913.

Copyright, 1914, by A. R. Elliott Publishing Company.

must have been to him a peculiar source of satisfaction to listen to the valuable presentations and discussions of such subjects as the functions of the cerebellum, to the elucidation of which he has so largely contributed.

It was a source of general regret that Sir William Gowers and Professor J. Déjerine were both prevented by illness from being present. Resolutions of sympathy and compliment regarding the former were unanimously adopted.

The name and fame of J. Hughlings Jackson, who was so recently removed by death, were recalled with affection and admiration in many personal conversations and in the speeches at the great dinners of the section and of the National Hospital staff, which were important features in the generous hospitality of the occasion. Dr. Thomas Buzzard, vigorous in mind and body at eighty-five years of age, presided at the National Hospital dinner.

Professor Heinrich Obersteiner, of Vienna, was greeted on all sides with the admiration and affection of his colleagues, many of whom had been former students at his celebrated laboratory in Vienna, greetings which he returned with cordial appreciation and to none more than to his former American pupils. Commanding figures in the section in neuropathology were Babinski, of Paris, von Monakow, of Zurich, and Petrén, the distinguished Swedish neurologist; among the Germans Liepmann, Rothmann, Anton, Saenger, Nonne, and Bruns were important contributors and striking personalities, while Italy was well represented by Bianchi, Donaggio and Bastianelli, and Austria-Hungary by Bárány, Frankl-Hochwart, von Eiselsberg, and Jendrassik. England's well deserved position of prominence in modern neurology was maintained and strengthened by the work of such men as Mott, Head, Batten, Purves Stewart, Kinnier Wilson, Collier, Holmes, and others, most of them members of the staff of the famous National Hospital. One of the most instructive meetings was that held in this hospital, where a series of cases of unusual interest and value were shown, and where moving picture demonstrations of nervous disease which held the close attention of an overflowing audience were given by Babinski and by Weisenburg.

At the meetings of the section and at the social board, Byrom Bramwell, courtly and dignified, represented the Edinburgh school, that seat of learning so dear to the hearts of graduates of the University of Pennsylvania, whose department of medicine was started on its long career of usefulness and distinction by graduates of the Scottish school.

France was well represented, not only as has been indicated in the discussions, but also by readers of independent papers which were without exception of a high order. Among these contributors were Claude, Léri, Guillain, Jumentié, and Barré. Claude also took an active part in one or two of the discussions. The papers of Foerster, well known for his early advocacy of rhizotomy for the treatment of spasticity, gave rise to an interesting discussion. Muskens, the Amsterdam neurologist, whose contributions on the sensory functions of the cortex are well known, had a good paper on the posterior longitudinal fasciculus. Auerbach and Grossmann presented a valuable contribution on the operative treatment of epilepsy.

America was well represented in the discussion of the myopathies by a paper of great merit from our colleague of this society, Dr. William G. Spiller. This contribution, which was received with much approving comment, presented the subject in a comprehensive and illuminating way. The contribution of Doctor Oppenheim, coreporter in this discussion, while brief, contained some points of particular interest and one or two somewhat novel, as, for instance, the inclusion in the subdivisions of myopathies, of myasthenia gravis and paralysis agitans.

Dr. Fred E. Batten, in discussing the papers of

Spiller and Oppenheim, presented, with lantern slide illustrations, valuable data and theoretical considerations. Dana, Sachs, Starr, and Elsberg, of New York, Schwab, of St. Louis, and Williams, of Washington, contributed to the section valuable clinical and clinicopathological papers; while A. R. Allen, of Philadelphia, gave the results of his elaborate investigations of the effects of impact in the production of changes in the spinal cord.

In addition to the symposium on the myopathies, four great discussions aroused and maintained the interest and enthusiasm of the section in neuropathology. These were The Symptoms of Cerebellar Disease and Their Significance, introduced by Dr. J. Babinski, Dr. A. Tournay, and Dr. Max Rothmann; Motor Aphasia, Anarthria, and Apraxia, with Dr. J. Déjerine and Dr. Hugo L. Liepmann as reporters; The Nature of the Condition Termed Parasyphilis, with Dr. F. W. Mott and Dr. Max Nonne as the first speakers; and The Treatment of Tumors of the Brain and Indications for Operation, the discussion being opened by Dr. Ludwig Bruns, Dr. Howard H. Tooth, Dr. Harvey Cushing, and Dr. A. von Eiselsberg.

In all of these discussions the number of those who took part was large and included such men as Thomas, Mendelssohn, and Holmes in the cerebellar symposium; von Monakow, Wilson, Bianchi, Claude, and Gutzman in that on aphasia; and Head, Sicard, and McIntosh in that on parasyphilis. The symposium on brain tumors was a joint affair of the section in neuropathology and the section in surgery, including, in addition to the reporters, Babinski, Burghard, Claude, Horsley, Jumentié, Krause, Macewen, de Martel, Paton, Pousseff, Starr, Sicard, Ternon, and Delagénière.

THE SYMPTOMS OF CEREBELLAR DISEASE AND THEIR SIGNIFICANCE.

While it is difficult justly to apportion the values of the different great discussions in the section in neuropathology, one of the most instructive and stimulating was that on the symptoms of cerebellar disease and their significance. It was in considerable part a presentation and discussion of the present status of cerebellar localization. In listening to the able papers of the reporters, Babinski and Tournay and Rothmann, and the discussion by those who followed them, my memory went back again to the American congress of twenty-five years before, at which the discussion of cerebral localization was a chief feature. It seemed to me that cerebellar localization as presented at the London congress was in about the same relative position of advance as cerebral localization at the congress in Washington in 1888. Prior to 1888, particularly as the result of work done during the twenty previous years, the doctrine of motor localization had been given a permanent standing in the physiology of the brain, and considerable subdivision of the motor zone authenticated by experimental and clinicopathological observation had been reached. The zone of speech had been expanded and subdivided so as to cover in a satisfactory manner the phenomena of aphasia for those who had given their allegiance and have since continued to hold to the so called classical views on aphasia. Much, however, remained to be done, not a little of which has been accomplished in the last twenty-five years. The motor zone now pushed forward to a region entirely or almost entirely in front of the central fissure, was then regarded by many as occupying a considerable area of the parietal lobe. The now well known parietal lobe symptom complex was just beginning to be developed. At present the symptomatology of this part of the brain is almost as clearly resolved as that of the motor region itself, and thanks to the impetus given by Marie and Liepmann, the study of aphasia has taken on new life and is leading neurologists into more critical methods of observing and thinking about the phenomena of speech. While much has been added to our knowledge of cerebellar localization during the last twenty-five years, these additions have been chiefly made in the last lustrum, or at least the last decade of this period.

In a paper like that here planned, which is intended to record for the most part simply impressions of the personnel and thoughts suggested by some of the important discussions, space cannot be taken for a detailed presentation of the data and views brought to the attention of members of the section in neuropathology of the congress. This would mean merely a more or less elaborate summary of contributions which in their entirety will no doubt soon hold the interested attention of neurologists everywhere. Fascinated by the wonderful neurological work of the congress, I find myself constantly tempted to reproduce and discuss it, but I shall try instead to limit myself to a mere glance at that which, it appears to me, would most attract the interest of a body like the Philadelphia Neurological Society, which since its organization has had for one of its objects keeping pace with real advances in neurology.

THE PAPER OF BABINSKI AND TOURNAY.

The subject of cerebellar symptoms was approached by Babinski and Tournay from a different angle from that of Rothmann. The former gave an admirable summary of the history of cerebellar research and observation, both experimental and clinicopathological, from the time of Flourens, in 1822, to the present. The parallel drawn by them between labyrinthine and cerebellar phenomena was a fitting introduction to the rest of their paper, which was taken up largely, first with the older classical cerebellar symptom complex, and secondly with the discussion of phenomena strictly cerebellar and, to some extent, capable of special localization; hypermetry and dysmetry, asynergy, adiadochokinesis, tremor, disturbances of writing, disorders of speech, asthenia, atony, cerebellar ataxia, and cerebellar catalepsy. Each of these subjects was treated in a masterly manner, and well it might be, as some of the symptoms and symptom complexes, were among the discoveries of Babinski himself.

As shown by Babinski and Tournay, the differentiation of labyrinthine and cerebellar phenomena is by no means easy, a few points, however, requiring particular attention. With regard to vertigo, observers differ as to its being a true cerebellar symptom, and the same difference of opinion is evident when nystagmus, so often tabulated among cerebellar symptoms, is under consideration. It is unquestionably true that both vertigo and nystagmus are often present in cerebellar lesions, but the topographical relations of portions of the cerebellum and the vestibular nerve apparatus, as well as the connections of the vestibular nuclei with the deep nuclei of the cerebellum, must not be forgotten. In a structural sense vertigo and nystagmus may be regarded as cerebellar symptoms, if caused by lesions involving within the cerebellum the tracts which unite the cerebellar nuclei with the nuclei of Deiters, Bechterew, and the triangular nucleus. The question for diagnosis would seem to be: Do lesions of any other portions of the cerebellum, of the cortex of any of its lobes or lobules, give vertigo or nystagmic phenomena?

Munk believes that these are not cerebellar symptoms, but that their occurrence in cerebellar lesions, experimental or otherwise, is due to involvement of neighboring parts, especially the labyrinth and vestibular apparatus. Horsley found that nystagmus and turning of the eyes were due to irritation of the white matter near the dentate nucleus and of the nucleus itself. Cocking of the head on one side, Batten believes, is due to a cerebellar lesion of the same side, while Horsley, from his experiments, is inclined to regard the symptom as vestibular. Babinski and Tournay make this point, that while some differences may be made out by close observation and investigation between classical labyrinthine and cerebellar symptoms, one must depend for exact diagnosis on symptoms which belong strictly to the cerebellum and are in nowise connected with lesion or disease of the labyrinth.

Among the most interesting parts of Babinski and Tournay's paper were those on asynergy and ataxia. The reporters recounted the well known previous observations of Babinski on asynergy, bilateral and unilateral, illustrating these by a discussion of the station and gait of the patients examined and by certain experiments with the limbs, especially the lower extremity of the one side, showing that in cerebellar cases, or some of them, distinct motor dissociation is present, for instance, between trunk and lower extremities in standing and walking, or between different sections of the lower extremities in movements of extension of the limb after the thigh has been flexed on the pelvis and the leg on the thigh. The uncertain or disorderly station and gait were shown to be largely due to the tendency of the trunk to hold back while the limbs advanced, or the reverse.

Babinski and Tournay's statements as regards cerebellar ataxia, or so called cerebellar ataxia, were almost as revolutionary in their way as those of Marie with regard to the classical idea of aphasia. They questioned the existence of a true cerebellar ataxia, as a pure symptom, at least. The uncertainty of station is, according to them, largely if not altogether a question of asynergy; hypermetry, tremor, and adiadochokinesis possibly entering into the phenomenon. Their comparison of the cerebellar station and gait with the station and gait of the tabetic ataxic was somewhat convincing in favor of their point of view. Cerebellar ataxia, if the views of Babinski and Tournay are accepted, is to be regarded simply as a disorder of equilibrium, static and dynamic, but not as representing a pure or segregated cerebellar phenomenon. Babinski in his exhibition of moving pictures showed a striking example of cerebellar catalepsy.

With regard to localization, it is instructive to observe that both sets of reporters in the cerebellar symposium, while speaking conservatively as to detail, were decidedly partial to the view that the cerebellum is not, as Luciani held, a homogeneous organ, but a collection of centres capable of being differentiated. Recognizing the cerebellum as, in a broad sense, entirely a motor organ, they held that the concatenation of facts is in favor of special localizations. The motor localization, however, differs in essential particulars from that of the representation in the motor zone of the cerebral cortex. It has relation essentially to a localization of limb, trunk, and head movements which are concerned with the maintenance of equilibrium. The centres are not representative of this or that distinct voluntary movement, simple or more or less complex, but of movements voluntary or semivoluntary, automatic or semiautomatic, which enable the human being to maintain the erect position and walk or perform the other motor functions in a regular and orderly manner; the centres are concerned in associated movements of the head and trunk, or limbs and trunk, or limbs and head and trunk, or different limbs, or different parts of the same limb. They are the centres in large part of reflex antagonistic movements.

The paper of Babinski and Tournay furnished in its data and in the manner in which principles were enunciated, a brilliant illustration of what can be accomplished by persistent and thoroughly conducted clinical work. More than once I have heard some of my Philadelphia confrères, and others of my countrymen not of Philadelphia, deplore what seemed to them to be the fact that clinical neurology had been so well worked that little of value and stimulating interest was left to be noted or discovered. Some of these deplorers of opportunity have indicated their belief that only in the anatomical, physiological, or pathological laboratory could we hope to get anything worth while investigating or considering. And this in the face of the fact that many of the greatest discoveries in neurology, as in other departments of medicine, have been the result of close clinical observation by men who had the keenness of intellect to discover new facts and new methods of interpreting old data.

A GENERAL GLANCE AT ROTHMANN'S PAPER.

Rothmann's report was largely concerned with the presentation of the results of experimentation on the cerebella of lower animals, especially dogs and monkeys or apes; although he did not neglect the important work in human pathology, and, among other things, especially directed attention to the remarkable investigations of Bárány. As a part of the cerebellar symposium Bárány gave a most instructive demonstration of caloric and rotatory tests for producing nystagmus and other phenomena, as those of dysmetry.

American neurologists reading the paper of Rothmann would be at first somewhat at a loss, unless they were not only acquainted with the embryology and comparative anatomy of the mammalian cerebellum but also had some knowledge of the new nomenclature introduced by Bolk. The anatomy of the human cerebellum, and most textbooks of comparative anatomy used by Americans, do not conform to the terminology adopted by Rothmann in his exposition. This paper, as I have already said, being intended merely to incorporate impressions and suggestions, it is desirable that the reader who wishes to be fully informed on the subject should not only study Rothmann's contribution to the Congress, but should consult directly the work of such investigators as Bolk, Van Rynberk, Elliot Smith, O. Charnock Bradley, and others.

A study of the embryology and comparative anatomy of the mammalian cerebellum will indeed contribute ideas of fundamental importance to the student of cerebellar functions, symptomatology, and localization. I find myself, after considering the contribution of Rothmann and the work of Bolk and others to which he refers, forced to believe that nearly all the published work on cerebellar disease by American and most foreign writers, especially by clinicians and clinicopathologists, is valueless in assisting one in arriving at conclusions as to cerebellar localization. This criticism is quite as applicable to my own work as to that of others.

In the first place, the attention of the neurologist in considering the localization of lesions in his cases of cerebellar disease, has usually been directed in the old fashioned way to a determination of whether or not the lesion present involves the lateral lobes or the vermis, and at the most, the anterior or posterior portions of these subdivisions of the cerebellum; the cerebellopontile angle, however, claiming often particular attention. The study of the mammalian cerebellum in animals below man, shows that the primary subdivision of this organ is not into a vermis and lateral lobes, but into an anterior and posterior lobe divided by a fissure called by most comparative anatomists the sulcus primarius or primary fissure, although known by other names. The posterior lobe in this subdivision becomes in the process of evolution, as man is approached, more and more complex, the anterior lobe retaining much of its original form and simplicity. As Bolk points out, the great distinction between the fully developed human brain and the brains of animals below man, even though close to him in evolutionary series, is in the extraordinary development of certain portions of this complicated posterior lobe, a development which even Bolk speaks of as relatively monstrous. In this fact, it seems to me, is to be found a clue to the physiology of a large part of the human cerebellum. This extraordinary development in the parts referred to must have relation to the work which the human cerebellum is called upon to do in maintaining the erect position of man and in enabling him to carry out the adjustments and adaptations of his head, trunk, and limbs, which give him a dominant position in skillful coordinations of every sort.

ROTHMANN'S RÉSUMÉ OF CEREBELLAR PHYSIOLOGICAL INVESTIGATIONS.

While highly important investigations in the domain of embryology and comparative anatomy were reported by Rothmann, he referred in addition to the results obtained by experimentation on the brains of lower mammals—dogs, monkeys, and apes—especially to extirpation experiments, like cortical encapsulation of a hemisphere, lobe, or lobule, although some determinations of value were reached by electrical irritation of such regions. Rothmann does not believe, as was held by Horsley and Clarke, that the cerebellar cortex is nonexcitable to the electrical current. It is more resistant to excitation, but does respond.

Rothmann called attention to the electrical investigations of Prus, also to the researches of Adamkiewicz and Pagano, all of which were in the direction of determining cerebellar centres for movements of the fore and hind limbs in dogs and centres for equilibrium.

Rothmann summarized the important investigations of Bolk and van Rynberk, which were in the form of both comparative anatomical observations and physiological experimentation, especially extirpation experiments. Bolk found in the crus primum of the ansiform lobe an unpaired (*unpaariges*) centre for the anterior extremities. By the "unpaired" centre, I take it is meant a centre which controls bilateral movements, that is, a centre on one side or in any position controlling the simultaneous movements of both extremities. In the crus secundum, Bolk found a similar or bilateral centre for both hind limbs or lower extremities. In the anterior lobe—that is, in the parts anterior to the sulcus primarius which corresponds to the preclival fissure in man—Bolk found coordinating centres for the movements of the eyes, tongue, muscles of mastication and expression. To the remaining portions of the mammalian brains studied and experimented upon, he assigned centres for the trunk musculature. Such remainder would seem to include the various portions of the inferior vermis and the so called paramedian lobe, that is, the lobes or lobules situated between the crus primum and the crus secundum and the vermis.

Van Rynberk confirmed in large part the investigations of Bolk, locating also a centre for the negation movement of the head in the anterior lobe. He also determined by extirpation of a portion of the crus primum of the ansiform lobe, a high raising of the front paw on the same side, or "goose step." Dysmetry of the hind paw in running was brought about by extirpation of the crus secundum or a portion of it. Extirpation of the paramedian lobe produced disturbance of the trunk muscles and rolling movements, while extirpation of the lobus simplex in connection with lesions of the crura prima caused very pronounced dysmetry of the forelegs. Other experimenters were summarized by Rothmann in a considerable list.

SOME PERSONAL OBSERVATIONS AND EXPERIMENTS BY ROTHMANN.

Turning now to the personal observations and experiments of Rothmann, these were of much interest and value, partly confirming the results obtained by Bolk, van Rynberk, Luna, and others and indicating to some extent the number of more or less exact and restricted localizations in the cerebellar cortex. Rothmann found, for instance, that extirpation of the lobus quadrangularis in a region corresponding to a part of the crus primum produced isolated disturbances of the foreleg of the same side, which exhibited themselves especially by lateral displacement of the limb and a dawdling or hesitating walk. Stripping the cortex of the part corresponding to the superior semilunar lobe, similar disturbance in movements of the hind leg resulted, while decapsulation of the inferior semilunar lobe, or its correspondent, and the paramedian lobe caused weakness of the lower regions of the trunk on the opposite side. Extirpation of the whole formatio vermicularis determined a bending and twisting or turning movement of the head toward the same side with disturbance of innervation of the ear of the same side, and a considerable weakness of the trunk muscles with intact position and movement of the extremities.

Bolk demonstrated that in lesions of those sections of the cortex that are turned under the fourth ventricle, especially of the lobus centralis, disorders of the jaw and larynx ensue. In destructions of this sort Katzenstein and Rothmann confirmed the abolition of barking, ataxic movements of the vocal cords limited in their excursion, and a pronounced weakness of the lower jaw which sometimes hangs directly down. Destruction of the cortex of the lobus medianus posterior was shown to lead likewise to an ataxia of the extremities, and to a disorder of the innervation of the trunk musculature which preponderatingly involves the lower section of the body.

According to Rothmann, injuries of the cortex of the vermis always cause combined disorders of the trunk and extremities; and statotonus, noted by Edinger, enters especially clearly into the phenomena.

From before backward in the cerebellar cortex, Rothmann held that there seemed to be represented definite sections of the body—eyes, head, face, jaws, tongue, neck, upper extremities, and lower extremities, and in the posterior median and paramedian regions,—the trunk.

More than this, Rothmann has apparently succeeded in isolating centres within the limb centres of the hemispheres (crus primum and crus secundum) so that in the foreleg region decortication of the lateral section allows displacement of the foreleg outward, decortication of the upper part causes bending and raising of the foreleg, and of the lower part, especially marked dropping of the foreleg. In the same way, in the hind leg region, decortication of the outer part causes displacement of the hind leg outward, of the inner part, displacement inward. These partial deficits, says Rothmann, are to be explained by an abolition of certain antagonistic reflexes of the muscles which normally serve to regulate the statotonus of the extremities concerned, called by Sherrington the proprioceptive reflexes.

I have given only a glance at some of the important data and conclusions in the wonderful résumé of Rothmann. His paper was a masterly one in its comprehensive plan, in its elaboration of details, and in his reasoning on the results of comparative anatomical research, physiological experimentation, and facts obtained through human pathology. To the highly important observations of Bárány, Rothmann, like Babinski and Tournay, gave particular attention, finding them corroborative of his own observations and those of Bolk, van Rynberk, and the others, to whose experimental work he calls attention.

DISCUSSION OF MOTOR APHASIA, ANARTHRIA, AND APRAXIA.

Among the strong personalities of the congress was Dr. Hugo Liepmann, of Berlin, who presented a paper on motor aphasia and apraxia of much interest and originality. Liepmann, with thrust and parry of assertion and subtle logic, unfolded his theory of apraxia as applied to the study of aphasia. The burden of his thesis was the similarity, if not identity, of apraxia and aphasia—apraxia in its different forms, sensorial, ideational, and motor, with the different so called varieties of aphasia. He held that asphasia could broadly be included in the definition of apraxia, apraxia being not simply the inability to do, but incapacity to understand the method of doing; in other words, the incapacity to apply knowledge purposively.

Illustrating apraxia in its simplest and most easily understood expression by limb movements, he showed, what is well known, that the apraxic becomes more or less incapacitated for using these movements intentionally or purposively. He attempts to light his candle with the match box; he is unable to brush and comb his hair because of his cerebral deficiency, or cannot use his hand for writing, although he knows what he wishes to write and in a certain sense how to write.

Since my return from the congress, I have examined a number of patients at the Philadelphia General Hospital—apraxic hemiplegics—these being sometimes aphasic and sometimes not—with the view of determining the presence or absence of apraxia and the degree of apraxia when this was present, also the special ways in which apraxia was most likely to be expressed. In one case of aphasia (motor or sensorimotor in type) the patient when asked to close his eyes closed his mouth, doing this repeatedly on different orders. When asked to put his hand on his shoulder he placed it on his head. Given a key to lock a door, he introduced this properly into the lock, but failed to turn it in the right direction. Another right hemiplegic and aphasic, when asked to place his left hand on his head, invariably raised the arm and held it flexed at the elbow a short distance from his head. Curiously, all of those examined on one occasion, five or six in number, combed the hair in proper manner, this test having been used because it was one of Liepmann's illustrations.

The ability of the patients to perform movements without the objects which were usually employed in carrying out such movements, was tested. The patient was asked, for instance, to show his interlocutor how to write his name in the air, that is, by movements of writing, also in a similar way to write easy words like "cat" or "sick" or "door," etc. After having been tested as to his ability to use a key in a door, he was requested to show how he would unlock a door. Similarly, experiments were made as to his ability to indicate how he would eat with a spoon or knife and fork, etc. The results showed inability or impairment of the power to do these purposive acts.

Liepmann, in his exposition of apraxia in its relations to motor aphasia and other forms of aphasia, starts with the fact that the human being is supplied at birth with a sensorimotor executive apparatus which he can use for many purposes, but which is not endowed with the attributes which will enable him to do the majority of purposive and especially skillful acts. To use Liepmann's own illustration, he says that if one had been raised by a she wolf, as Romulus and Remus were, he would have such limb movements, and movements and abilities in general as could be learned from the she wolf. He would not, like the children raised in human environment, acquire the ability to perform skillful and varied purposive movements in accordance with the higher human acquisitions. In other words, the child adds to his attributes and abilities as the result of imitation of his fellows and of instruction by them. Liepmann had little or nothing to say about inheritance in this relation, but probably assumes an inherited capacity for the acquisition of the higher purposive movements in the human being.

With regard to localization of so called centres, cortical and subcortical tracts in the cerebral zone of speech, Liepmann had not very much to say. He seemed to accept, to a considerable extent if not completely, the classical ideas and data as to such localizations. He argued, however, in favor of the close connection, anatomically as well as physiologically, of these centres with the executive sensorimotor apparatus. The mnestic centres for hand movements, for instance, he holds are before, behind, and in the Rolandic region, and similarly, the speech centre of Broca reaches over into the executive laryngoglossal centres for movement. Owing to the anatomical connection between the centres, mnestic and executive, one of these regions, cortical or subcortical, is rarely injured without the other being involved. Hence, we have the admixture of aphasia with anarthric disturbances. He expressed simply in other words the doctrine of neural (cerebral) mechanisms or that of the diaschisis of von Monakow.

Professor Déjerine, because of illness, was not present at the congress, a fact which was much regretted. He sent, however, his contribution on the subject of motor aphasia, which was read at the section by Jumentié. This paper was a careful and convincing restatement of what might be called the classical view of the subject of motor aphasia, the arguments and many of the data used being the same as those employed by Déjerine in his discussions with Marie, in 1908, after the latter expounded his new views regarding aphasia and mental deficit and challenged the part played by Broca's convolution in the function of speech. To his data and arguments as hitherto expressed, Déjerine had added some facts and opinions gleaned from the recent discussions of aphasia.

The first part of Déjerine's essay was an historical résumé of the localization of the function of motor speech in the anterior part of the left hemisphere, especially in the hinder part of the left third frontal convolution. Well known names, like those of Bouillaud, Andral, Broca, Gairdner, Hughlings Jackson, Bateman, Falret, Trousseau, Charcot, Wernicke, Ladame, etc., naturally figured in this part of his presentation. New additional names were added, more especially as the result of the discussions of the subject during the last five or six years.

Taking up anarthria and motor aphasia in connection with the views of Marie and Moutier, his paper contributed an admirable discussion of this subject. The early and first accepted idea of anarthria, as a paralytic affection of speech due to bulbar disease, was dwelt upon, the speaker showing how, as time progressed, true anarthria was seen to be also a pseudobulbar affection due to disease, especially bilateral disease of the geniculate bundles of the internal capsule. He believed that the term anarthria should be restricted to these paralytic affections of speech, bulbar or pseudobulbar. An interesting point was that made with regard to Moutier's enlarged definition of anarthria so as to make it accord with the views of Marie. According to Moutier, in anarthria as recognized by Marie and himself, the patient did not know how to speak, whereas in the anarthria of Kussmaul and manv others, he could not speak. Déjerine, regarding this new definition, says that this accepted, his own views would not differ materially from those of Moutier, for the props fall from beneath the Marie contention. In true motor aphasia the difficulty is just this, that the patient does not know how to speak, that although his organs of enunciation and articulation may be in good order, it is impossible for him to use them for the purpose of language.

Déjerine discussed the part played by the lenticula, especially the putamen of the lenticula, in the affections of speech, referring to Broadbent, Mingazzini, Mills and Spiller, and others in connection with this subject. On the whole, he argued against the lenticula playing any real part in speech, holding that in most cases of bilateral lesion of the lenticula, lesions were also present in the internal capsule.

Déjerine also discussed the existence of a speech projection tract separate from the geniculate bundle and somewhat anterior to it. He opposed the views of Brissaud, Raymond, and others, who held to the belief in such separate tract, contending that the cases cited in support of it could not be satisfactorily maintained as corroborated. In connection with this subject, he discussed also the existence of bundles concerned with the transmission of speech impulses, such as have been suggested by the pathological observations of von Monakow.

While paying a tribute to Liepmann's researches on apraxia, Déjerine felt that we cannot fully accept the idea that the phenomena of aphasia could all be translated into terms of apraxia. Apraxics might be aphasic, aphasics might be apraxic, but aphasia and apraxia were not identical; there was more than apraxia in aphasia.

Déjerine's presentation of the question of the part played by Broca's convolution in the function of speech was as creditable as it was convincing. He showed that the weight of reported cases favored this view, throwing the burden of proof on those who had used presumably opposing data.

THE DISCUSSION OF PARASYPHILIS.

Time can be taken to say only a few words about the discussion of parasyphilis, a subject now attracting widespread attention throughout the medical world. A vein was struck in this discussion different from that in either cerebellar localization or aphasia and apraxia, for vital questions of prognosis and treatment were pertinent to the presentation and consideration of the subject.

Nonne, Mott, and Head were prominent personalities in this symposium—three men who seemed to me, although having different characteristics, to possess powers which have made them justly famous in their special lines of work during recent years. Nonne impressed one as a hard working, clear seeing, and quiet enthusiast, Mott a thoroughgoing, logical British type.

Regarding Nonne's presentation, I shall simply glance at a few points, those which appealed most to me or which I remember best. One of these was the parallel which he drew between affections of the brain and cord and peripheral nerves due to the influence of the trypanosoma, and such so called syphilitic diseases as tabes and general paresis. His reason for doing this was evident, namely, to show

how microorganisms with their poisons of a different sort from Spirochæta pallida may attack the substance of the nervous system and produce effects like those due to the acknowledged syphilitic organism. The general tendency of his report was to strike down the artificial barriers which have so long been set between direct syphilis and indirect, that is, parasyphilis or metasyphilis. Clinical and pathological observations were brought to this task in almost equal number—abortive and irregular types of paresis and tabes; findings in membranes, vessels, nerve roots, and in both spinal and cerebral substance in the same and different cases, etc.

That tabes and paresis have been demonstrated to result in cases of infection without primary lesion, as from penetration by an infected needle, and that parasyphilis can probably be produced through spermatozoic infection without the necessity of cutaneous, mucomembranous, or other lesions, were among Nonne's data of especial interest. An important part of Nonne's report was his discussion, largely antagonistic, of the views of those who hold to neuropathic predisposition in cases of syphilitic infection which result in parasyphilitic disease. Most suggestive was his consideration of the view that possibly different biological forms of the spirochæta may be influential in causing direct syphilis of the nervous system on the one hand, and parasyphilis on the other. Toward the close of his paper he made the significant admission that, as he expressed it in good American fashion, "much is still in the air."

Mott's remarks and suggestions as to the parts played by high and low aortitis (topographically speaking) in the development of paresis and tabes were among the interesting points in his paper.

DISCUSSION ON THE TREATMENT OF TUMORS OF THE BRAIN, AND THE INDICATIONS FOR OPERATION.

A wonderful array of surgeons and neurologists was lined up in the discussion of the surgical treatment of brain tumors; Krause, von Eiselsberg, Cushing, Bruns, and Tooth were among the number. The personality and methods of presentation of von Eiselsberg were charming and convincing. I had the opportunity of talking with him outside the meetings of the section as well as listening to him in the discussion, and was much impressed with the feeling that he was one of the most distinguished of the leaders in modern surgery. It was much to be regretted that Philadelphia surgery was not represented in this discussion, as I feel that, with the exception of von Eiselsberg and one or two others, American neurological surgery is somewhat ahead of that of Great Britain and the continent in matters of technic, in special methods of operating, and in the results obtained. Many of the foreign surgeons, so far as I could make out, still adhere to the old method of using the trephine and rongeur rather than the osteoplastic operation performed with the Hudson trephine and electrical engine and accessories, although regarding this I may not be altogether correct. I am not sure that the methods employed did not partially account for the somewhat unfavorable results of some of the operations reported.

My mind was in antagonism to several of the suggestions offered in this discussion, for instance, to one that practically all cranial operations for tumor should take at least four hours, that is, that the operation should of necessity be slowed down with unusual care until it covered a period of this length. My own view from much interested looking on during brain tumor operations is that every such operation should be performed as rapidly as possible, qualified only by the opinion that safety should not be sacrificed to extra speed.

1909 Chestnut Street.

Reprinted from the New York Medical Journal for May 16, 1914.

APRAXIA IN RELATION TO APHASIA.*

By John H. W. Rhein, M. D.,
Philadelphia,

Professor of Diseases of the Mind and Nervous System, Philadelphia Polyclinic and College for Graduates in Medicine; Neurologist, Howard Hospital; Physician, Home for Incurables.

The relation that apraxia holds to the subject of aphasia has been hitherto exemplified in connection with certain forms of aphasia only, namely, motor aphasia, agraphia, amimia, and amusia, but the recent work of Liepmann shows that not only motor aphasia but every form of expressive aphasia and apraxia are similar. Before reviewing Liepmann's (1) ideas without which no discussion of the relation of apraxia to aphasia could be complete, it will be necessary to refer to some of the features of apraxia in order to show clearly the bearing one has on the other.

Apraxia is an inability to perform purposeful movements, while at the same time the object and its use are recognized and the limbs can be readily moved, in other words, are not paralyzed. It is usually unilateral, affects simple movements, interferes with imitation of movements, and gives rise to amorphous instead of purposeful movements. There is recognized also an ideatory apraxia which affects mainly the manipulation of objects, for example, a cigar is rubbed against the match box instead of the match, in an effort to light the cigar. This is called association apraxia by Bonhoffer. There is then the *Glied-kinetic* apraxia of Kleist in which movements which arise from practice, such as writing, drawing, and knitting, are implicated. This is the cortical apraxia of Heil-

---

*Read before the Philadelphia Neurological Society, February 27, 1914.

Copyright, 1914, by A. R. Elliott Publishing Company.

bronner. Finally, there is amnesic apraxia in which the subject forgets, on command or invitation, how to perform certain purposeful movements.

While aphasia may or may not accompany apraxia they are often associated in the same case (Liepmann (2), von Stauffenburg (3), Pfeifer (4), d'Hollander (5), Kroll (6), and others). This association is not difficult to understand when we reflect that the lesions causing aphasia are to be found in regions closely related to those causing apraxia. The lesion in apraxia is found most commonly either in the gyrus supramarginalis, in the frontal lobe, in the corpus callosum and supracapsular areas, or, finally, in various locations.

The association of apraxia with aphasia at first sight is more apparent in relation to motor aphasia. Motor aphasia has been called apraxia of the speech muscles by Nikitin (7). Liepmann (8) said in an earlier communication that in a broad sense, expressive aphasia is apraxia, consisting of an inability to utter words, while the tongue, palate, mouth, and vocal cord musculature is intact. In motor aphasia there is an inability to use the tongue, lips, soft palate, and vocal cords, while at the same time these muscles are not paralyzed. The muscles can be moved, but the subject cannot awaken the proper movement memories to produce speech. In apraxia there is an inability to use the arm for purposeful movements, yet there is complete power to move the arm muscles. Aphasia is to the speech musculature what apraxia is to the limb musculature.

The idea of a word is made up of auditory images of the sound of words, visual images of writing and printed words, speech kinesthetic images, and, finally, graphic kinesthetic images (Wilson). Speech itself is acquired by a storing up of auditory images. On the other hand, purposeful movements depend largely upon the storing up of visual and kinesthetic memories. If the auditory memories or images cannot be recalled we have aphasia; if the visual and kinesthetic memories cannot be recalled we have apraxia. Liepmann has gone further and considers, not only motor aphasia, but every expressive aphasic disturbance of speech and writing essentially similar to apraxia. His views must be given in considerable detail in order to present the subject in a clear manner.

He differentiates three principal forms of apraxia, namely, 1, ideational apraxia after Pick; 2, limb kinetic apraxia, in which limb kinetic engramms are lost; 3, ideokinetic apraxia, in which the association between the ideation and the limb kinetic is disturbed. There is, he considers, no reason to limit the conception of apraxia to visible effects of movements.

The executive apparatus (sensomotorium) is not sufficient for the function of life, and has superimposed kinesthetic memory ("the memory for the complex of sensations which movement itself leaves behind") and the innervation complexes existing as engramms of the nerve substance, which together he calls a kinetic memory or a memory one acquires by practice for certain forms of movements. But this is not sufficient, as it is only for simple and much practised movements and is not adequate for the daily requirements. These (memory and innervation or limb kinematic) then must be brought into extensive cooperation with the whole brain. Hence we then have limb kinetic memory and the connection of this with the remaining possession of memory by which unaccustomed series of movements are possible and there is a capacity to use "the limbs according to purposive ideas."

Movements of the extremities is a matter of a "sketch of movement whose base is a space time series (*Bewegungsformel*) in a covering of a sensory quality, generally a visual covering. This he calls the ideational sketch which tells one which limbs and parts of limbs are to be moved and their order and "what ways they have to take." The availability of the limbs for purposes, therefore, are dependent on "1, the ideational sketch; 2, limb kinetic memory; and, 3, regular correlation between the two." This also holds good for the laryngopharyngoglossolabial muscles, whose visible effect is mimic movements, and audible effect speech.

Speech has its executive apparatus, a limb kinetic faculty and a kinesthetic engramm (the formerly overrated so called "representation of the movements of speech"). The kinetic possession of words is insufficient to maintain talking, and ideation for the word and sentence requires instead of time-space-visual directives as in movements, time-auditory directives.

With most people the motor word is revised by the auditory component of the word, not directly by the idea, and when these are impaired (sensoric aphasia), "the limb kinetic possession of letters and syllables is retained, but the succession, the choice of the suitable letters and syllables is defective" (paraphasia).

To produce speech properly the acoustic sketch must influence the kinematic of the speech muscles, as, in apraxia of the limbs, the visual sketch influences the kinematic of the extremities. This paraphasia occurs if the acoustic sketch or its connections with the kinetic sphere is defective and has a "similar habitus sometimes like the ideational and sometimes like the ideokinetic defective reactions of the apractic patient."

In apractic patients, the ideational defective reactions are seen in "perseveration, omission, duplication, anticipation of something later, glancing off to something related, becoming directed away to accidental, sensory impression, a mixture of perseveration with something newly intended, etc., briefly a confusion of motor intact partial actions."

In paraphasia (sensory aphasia) there is "perseveration to earlier syllables, anticipation of later syllables, sliding off into words related in sound and sense, fusion of parts of suitable words with those of similar sounds and similar sense and those of persevering words, connections with parts of words which are accidentally awakened by sensory impression or associations." Here there is "an analogy of exchange of movements (*Bewegungsverwechslung*) of the ideokinetic patient in *verbal* paraphasia."

Amnesic aphasia (difficulty in finding words) generally due to a difficulty in reviving acoustic engramms (slight injury to Wernicke's zone) corresponds to amnesic apraxia (nonoccurrence of movement). There is an ideokinetic paraphasia also, as when spontaneous speech is difficult (difficult awakening of the kinetic words). The kinetic engramms are retained, as is seen in preservation of repetition, but strong acoustic stimuli are required to render them active. But as acoustic memory possession for words differs from visual memory possession for movements, and as there are "many other differences in the mechanism of speech and other movements, one is forbidden from carrying the analogy of the two to an extreme, i. e., distributing all aphasic disturbances among forms of apraxia."

Liepmann wished principally to show that, physiologically and pathologically, the expressive aphasias belong to the sphere of apraxia. In considering the question of localization he stated that the limb kinetic engramms of the hand are located in front of, behind, and in the central convolutions, and that the engramms of the speech movements stretch over from Broca's region to the opercular part of the anterior central convolutions, so that the mnesic and executive apparatus partially cover one another.

Liepmann has shown that the left side of the brain preponderates for praxia as for functions of speech. The lack of independence in the right brain is shown particularly in the inability to perform movements *without* an object. Feeling and seeing the object admit of the movement. He calls these objectless movements free movements, and he considers then that the right hemisphere is dependent for these on the left. Speech is a free objectless movement, and the mouth and tongue produce words only by the help of the acoustic kinetic image, while the hand movements of beckoning or threatening (objectless movements) require the optic kinetic image. "The inferiority of the right hemisphere for phasia and praxia can be referred to one and the same deficiency, i. e., that of not directing movement without object." The result, he considers, supports biologically the essential similarity of phasia and praxia which he outlined.

The similarity of apraxia and agraphia, the latter being a form of aphasia, is thus also explained. To us the relation seems particularly apparent with that form of agraphia which is called isolated agraphia (Wernicke) or pure motor agraphia (Pitres and Déjerine).

The existence of isolated agraphia has been known for a long time, but in view of the recent studies of apraxia it takes on a different significance. Writing results from the awakening of graphic kinesthetic memories lodged in the auditory image centre, the visual image centre, the speech kinesthetic centre, and the centre for ideas of objects. As Wilson (9) has put it, if the afferent apparatus is perfect and the patient is, notwithstanding this, unable to revive graphic kinesthetic images, he is agraphic without paralysis. He has apraxia of the movements of writing. It was Mass (10) in 1898, who was the first to describe the relation existing between agraphia and apraxia. Later Liepmann and Mass (11) described a second case of left sided agraphia and apraxia with right sided palsy. The difficulty in writing in this case they looked upon as motor apraxia and as not being related to the disturbance of the act of writing dependent upon internal speech. The lesion was in the corpus callosum, leaving the cortex intact in the frontal, parietal, and occipital regions. The dyspraxia was due to the robbing of the right arm centre of the control of the left arm centre, by the foci which implicated the corpus callosum. Kroll's (6) view was that the right supramarginal gyrus, where are stored the kinesthetic memories for the left arm, was separated from the left sensomotorium by the lesion. Hartmann (12) and Lorenz's (13) cases are also examples of dyspraxia due to destruction of the corpus callosum. In Heilbronner's (14) case, "hacks" were made with a pen instead of letters. Bonhoffer (15) reported a case in which the patient was unable to write certain letters, though he knew them, but instead produced characterless figures. He believed this was apractic agraphia.

Liepmann, in his earlier paper, looked upon the right sided agraphia in his case as an expression of apraxia. He could write with the left hand, showing that internal speech was preserved, but he was unable to send impulses to the hand centre in the left cerebrum. In connection with this subject, the case of Meyer's is interesting, being one of apractic agraphia in a right handed person, with paralysis of the left arm and leg and preservation of speech and the ability to write. Bychowski (16) believed that the difficulty in writing in his case was the result of a disturbance of manipulation, that his patient suffered from apraxia of the faculty of writing words.

The relation of apraxia to instrumental amusia is apparent, indeed instrumental amusia is apraxia. Pick (17), in discussing the relation of ideatory apraxia and instrumental amusia, cited some interesting cases from the literature. In Charcot's patient, a trombone player, all the motor memories were intact, the memories for associated movements of the hand and mouth necessary to play his instrument having been lost.

In Starr's case of pure motor aphasia and agraphia without paralysis, the patient could read and enjoy music, but was not able to play the piano. Kast's patient held his violin like a piece of wood. There have been analogous cases in violin players (Oppenheim). Instrumental amusia in Pick's case, he believed, was an apractic symptom due to a functional disturbance, the amusia resulting from the lapse of the implicated kinesthetic memories. Pick further cites the cases of Donath, who could only play one piece, each effort to play other pieces resulting in the performance of the same piece. In Würtzen's case there was complete instrumental amusia of the right hand which was not palsied nor agraphic, while the left hand was intact.

According to the studies of Edren, the centre which, when injured, causes amusia, is situated in the first and second temporal convolutions, and if this is true the relation anatomically to apraxia is evident. Pick looked upon apraxia in certain cases as independent of sensory or motor disturbances, and as representing an ideomotor trouble, and it is, in his opinion, a functional disturbance in which there is a weakness of the memories for purposeful movements. Finally amimia, the loss of expression in gesture or mimicry may be looked upon as apraxia.

Apraxia may occur also in cases of mental disease just as aphasia occurs in mental disease. It has been described in senile dementia by Marcuse (18), Drommard and Pascal (19); in general paralysis by Abraham (20), and Lewandowski (21); in senile cerebral atrophy by Stransky (22); in dementia præcox by Drommard; in mental confusion, the amentia of Meynert; and in confusional states in general, such as posthysterical and post-epileptic conditions, by Pick, and in the delirium of alcoholism by Bonhoffer. Van der Vloet (23), as a result of a study of fourteen cases, concluded that dementia and apraxia are independent of each other.

Marie (24) expressed the view that apraxia is the result of an intellectual deficit, and that apraxia is really an aphasia of feeble intensity. He believed that the order is not understood, nor does the patient have an exact idea of the gesture which corresponds to the given order. On the other hand, according to Liepmann, the mental deficit is the result of the lesion causing the apraxia and not the cause itself.

My own view is that a certain degree of intellectual deficit exists in these cases. The patient does not always recognize the incorrect result, in fact, believes that he is performing the act properly, when in reality he is apractic in his movements. This may be purely a psychic deficit or it may be agnosia or, what is more likely, a combination of the two.

REFERENCES:

1. LIEPMANN: *Monatschrift für Psychologie und Neurologie.* xxxv. 2. IDEM: *Berliner klinische Wochenschrift,* xlix, p. 2378, 1912. 3. VON STAUFFENBURG: *Zeitschrift für die gesamte Neurologie und Psychologie,* v, p. 434, 1911. 4. PFEIFER: *Journal für Psychologie und Neurologie,* xviii, p. 23, 1911. 5. D'HOLLANDER: Encephale, 1912, i, p. 506. 6. KROLL: *Zeitschrift für die Neurologie,* ii, p. 315, 1910. 7. NIKITIN: *Allgemeine Wiener medizinische Zeitung,* li, p. 105, 1911. 8. LIEPMANN: *Monatschrift für Psychiatrie und Neurologie,* xvii and xix. 9. WILSON: *Brain,* xxxi, i, p. 164, 1908. 10. MASS: In Bychowski (*Vide* 16). 11. LIEPMANN and MASS: *Journal für Psychologie und Neurologie,* p. 214, 1907. 12. HARTMANN: *Monatschrift für Psychiatrie und Neurologie,* p. 112, 1907. 13. LORENZ: *Zeitschrift für Psychiatrie,* lxiv, 1907. 14. HEILBRONNER: *Handbuch der Neurologie,* ii, p. 082, 1910. 15. BONHOFFER: *Archiv für Psychiatrie,* xxxvii, p. 800, 1903. 16. BYCHOWSKI: *Monatschrift für Psychiatrie und Neurologie,* xxv, p. 1, 1909. 17. PICK, A.: *Studien über motor Apraxie,* 1905. 18. MARCUSE: *Centralblatt für Nervenheilkunde,* p. 737, 1904. 19. DROMMARD and PASCAL: *Presse médicale,* xvii, p. 275, 1909. 20. ABRAHAM: *Allgemeine Zeitschrift für Psychiatrie.* p. 502, 1904. 21. LEWANDOWSKI: *Centralblatt für Nervenheilkunde,* p. 705, 1905. 22. STRANSKY: In Wilson. 23. VAN DER VLOET: *Journal de neurologie,* p. 589, 1906. 24. MARIE: *Presse médicale,* xxvi, p. 242, 1906.

1732 Pine Street.

235

PRESIDENTIAL ADDRESS BEFORE THE AMERICAN PSYCHOPATHOLOGICAL ASSOCIATION

BY ALFRED REGINALD ALLEN, M.D.

(PHILADELPHIA, PA.)

Albany, N. Y, May 6, 1914

* * *

REPRINTED FROM

THE JOURNAL OF ABNORMAL PSYCHOLOGY

BOSTON

October–November, 1914

PRESIDENTIAL ADDRESS BEFORE THE AMERICAN PSYCHOPATHOLOGICAL ASSOCIATION

Albany, N. Y., May 6, 1914

BY ALFRED REGINALD ALLEN, M.D. (PHILADELPHIA, PA.)

WHEN I contemplate the membership of this most active association, when I realize the progressive character and the amount of the work which has appeared in the few short years of its life, an abashment I fain would repress forces its way into my consciousness and I confess inwardly and to you that I am not worthy of the honor you conferred upon me when you made me your president.

Therefore in opening this, the fifth annual meeting of the American Psychopathological Association, I bow to you, my betters, and with feelings of gratitude and admiration I say: — may I learn much.

A retrospect of the work of the past year in psychopathology in the United States and Canada would, I am sure, make clear the fact that the vast proportion of what I may term dynamic psychopathology has come from members of this association. An analysis of this material would be instructive and interesting but hardly the best use of what I promise you will be a short time.

Of all the interesting episodes in psychopathology of recent occurrence, I think I am safe in saying that none is of more interest to us, nor is any likely to have a more far-reaching affect, than the Freud-Jung controversy. This great parting of the ways strikes many varying chords, depending on the attitude of the man giving it attention. There are unbending ultra-partizans of both sides, to whom the follower of the opposite faction is a hissing and reproach. There are many who have received the teaching of the Vienna school with respectful attention and, who, although not able to subscribe to all the tenets, yet have found much food for thought and no small amount of help therefrom. This class divides naturally into those who view the dispute as a calamity, to whom the veil of the temple is rent in twain; and into those who see in the *internecine mêlée* the potential for a reconstruction, in which only the tried and proved masonry would find lasting place in a fabric which should the better withstand the storm and waves of unreason, and in the end prove that it be not built upon the sand. Lastly, there are many of the type of the Merry Andrew, who have already hailed the rift with delight as a productive field for what they doubtless consider true wit. These and their vapid criticisms need detain only those with petrified sense of humor. Their opinions have the distinction of being likened to the "crackling of thorns" in a certain classic.

It is my purpose to refer to a few of the chief points at issue between these two schools of thought, and to try to see whether the greatest and most fundamental ideas have been abandoned by either one.

The point of contention which I shall first mention is the question of the sexual theory and infantile sexuality. Abraham, it seems to me, has made a good point (*Internat. Zietschrift für ärztliche Psychoanalyse*, page 72, January, 1914) in his criticism of Jung's identification of sexuality with the instinct of species preservation, when he says, "*Trieb der Arterhaltung nichts ist als eine teleologische Fiktion.*" His idea is that because all phenomena of developing sexuality are links in a chain which ultimately leads to the sexual act and through this to child-birth, the essence of species preservation, this is no reason for arguing that this preservation is the final terminus and that all along the line the various phenomena have been motivated with this one end in view. He rather takes the position that child-bearing is altogether a by-phenomenon, which of course incidentally preserves the species, but that the motive from earliest inception to development is one of "*Lustgewinnung.*"

Jung departs somewhat from the concept of Freud concerning the act of sucking at the breast, and makes the statement that sucking is not sexual pleasure but nourishment pleasure. It seems to me that here Jung has been quite as dogmatic as has Freud. If there have been one characteristic in the teaching of the Vienna school which has struck the average man as incongruous, it is a tendency to state a theory as a proven fact. This is often apparent in writings dealing with the interpretation of dreams where the manifest content is stated, and immediately after the latent content given without any connecting link to show how the interpretation was reached. Many of the opponents of Freud have the idea that there is a certain formulary by which the psychoanalysts think they are able to check off the symbolisms of the manifest content. Unfortunately, in the work of some men this reproach is not altogether without reason. Likewise, in the explanation of the objective by-phenomena of sucking when proof is requested as to the sexual content of this act, one is asked to accept an argument which to my way of thinking carries but little weight.

Now, in regard to this act of sucking at the breast, why is it necessary to determine a prototype to which all infants must measure? What valid reason can be advanced against the plea for individuality in the infant! Surely none at this day contends that psychic complexes are all the result of synthesis, on the part of the child, of exogenous factors, and that the different pictures spring from an identical psychic level.

I take it the brief may be defended that there are true, infantile, erogenous zones; that the delimitations are as varied as the physical characteristics; that there is a process of what may well be termed convergence; and finally, that the rate of this process may vary greatly.

Freud's concept as to sexual pleasure in the act of sucking presents no obstacles to hinder acceptance if it be advanced as possibility rather than dogma. But on the other hand, to speak of the pleasure of nutrition seems to me a poor way of indicating a simple biological fact, to wit: the relief due to the temporary suspension of the chemical stimuli caused by hunger, which stimuli are of a painful character in the adult, and are surely no less pleasant in the infant where metabolic activity is more pronounced.

Yet who can affirm that there be no admixture of sexual pleasure due to *co-existent* erogenous zone stimulation? I admit that to me the sight of the baby at the end of the sucking act presenting a relaxed, satisfied picture and tending to sleep is a weak argument on which to support a theory of sexuality of the act of sucking. The dog-breeder has possibly observed a much more pertinent and interesting phenomenon while watching a litter of puppies suckling. A puppy at times will not only make use of the purely utilitarian thrusting forward of the head, which movement accelerates the flow of milk, but occasionally a most stereotyped and correct pelvic action will be demonstrated, which is an exact replica of the movement of copulation.

Now, in this relation it becomes evident that there is far from being harmony among those interested in psychoanalysis as to the breadth of the term *sexual* when applied to early child life. Jung's intimation that there are those who mean by the term sexuality "the fully developed function" may be true concerning the laity, but hardly can obtain among psychologists. But following this thought Jung does not make himself at all clear to me as to exactly how he means to interpret the term. I am perfectly willing to admit that this lack of appreciation is due to a short-coming on my part, an explanation which I regret to say is heard frequently from members of the psychoanalytic movement, when one does not understand their point of view. But I am at a loss to see how it is possible for Jung to say first that " . . . *der wichtige Trieb der Arterhaltung schon in frühester Jugend keimend sich zu entfalten beginnt,* . . . " from the context clearly indicating sexuality, and then to follow this in the next paragraph with the statement: " . . . *solle man die andeutenden und vorbereitenden Phänomene der infantilen Zeit lieber nicht als sexuelle bezeichnen.*"

At the meeting of the association last year there was a statement made in the discussion of one of the papers, which statement was evidently removed from the report of the transactions, as I fail to find it in the printed report in the JOURNAL OF ABNORMAL PSYCHOLOGY. This statement was actuated by some interpretation of the term sexuality, and was to the effect that when used in the sense of Freud it was in no sense divorced from a gross and animal significance, but was the same concept one would get if he asked the first man in the street what he understood by the term. I do not see how any one holding this meaning of the word could other than reject the teaching of the Vienna school concerning the psychology of infancy. But I do not think there are many who accept such an interpretation.

In the early development of the human embryo it has been abundantly proven that physically ontogeny reproduces phylogeny. We find in early stages an organ, let us say the kidney, represented in part by the mesonephros. There is no mesonephros in the new-born baby, but on the other hand a well-differentiated kidney. Nevertheless, this mesonephros was the anlage of a part of the kidney, was the true representative of part of the kidney, and because of the latter differentiation into kidney there is no possible right to deny to it its identity in the scheme of nature. Moreover, when we look at some of the lower forms of life we find this mesonephros not a transition product but an end result with the function of an organ of elimination.

Now, for mesonephros read *an elementary, non-differentiated feeling of pleasure* from some stimulation; for kidney read *the sharp-cut, well-developed libido of the adult*,— and here I am willing to be as specific as can be and state that I refer to the psycho-physical gratification of sexual desire by the sexual act. In other words there is no adequate reason for rejecting the proposition that psychically ontogeny reproduces phylogeny. Only let it be understood that this is a theorem after which we cannot as yet write *quod erat demonstrandum*. Also understand that in the contemplation of the physical phenomena above, we have dealt with embryonic life, but in regard to the psychic phenomena, the development I have indicated — the differentiation from the elementary anlage to the end product — I assume to take place altogether after birth.

Jung approaches the libido theory from the polymorphic perverse sexuality of infants. In this regard his likening of the changes in the quality of sexuality, from the heterologous to homologous, for instance, to the transformation of one type of energy to another is ingenious. But if he mean to infer that the psychology of Freud precludes such a concept, and to liken the Freud concept to the pre-Mayer stage of physics, I hardly think he has been fair to Freud. It seems to me that Jung's objection to Freud's concept of libido arises largely from an unwillingness to accept a broad meaning for the term sexuality. On this point I realize that the Vienna school itself is not altogether in unison. Taking a narrow meaning of the term sexuality I can well see Jung's objection. But accepting, as above, that sexuality is of broad significance, is elementary and non-differentiated, I see no incongruity in the concept libido as held by Freud, nor is it apparent that this concept clashes with Jung's ideas, except in so far as Jung advances the time of life for the manifestations of libido. In other words, the difference is rather quantitative than qualitative.

Jung makes this latter point clear in his classification of the three phases of life, in the first stage of which,—"*vorsexuelle Stufe*," he speaks of the libido of nutrition and pictures its "*Wanderung*" (march), by which he means its passage from the nutritional to the sexual realm. But in this connection it becomes evident that the difference between the schools is one chiefly of definition, particularly when it comes to the discussion of infantile perversity.

The rather patronizing air which Jung adopts in dealing with Freud's following what he, Jung, considers a false way in the traumatic theory is really not worth while mentioning, except that it is unfortunately calculated to stir up bitter feeling.

Jung's method of dealing with the unconscious seems to be more of an exposé of the reasons why the opponents take issue with the psychoanalytic school rather than an attempt to portray a mental state. His analogy of the bridge builder might almost be taken to indicate that the uninformed had no business requiring any demonstration or explanation simply because the psychoanalyst had pronounced things to be so ordered. This is rather after the order of καὶ ὁ λόγος ἦν πρὸς τὸν θεόν, καὶ θεὸς ἦν ὁ λόγος and if followed to its logical conclusion would lead us to the dictum that no one who was not following the practice of psychoanalysis had any right to expect to understand its *rationale*. This attitude is at times taken by psychoanalysts who are pressed for explanations of certain phenomena, and it never fails to make an unfortunate impression.

Jung accentuates the idea of regression of the libido to an extent that the part played by fixation is well nigh eclipsed. Now, the concept regression as well as fixation is quite indigenous to the school of Freud, and, therefore, as there is no fundamental mechanism questioned by either Jung or Freud it would seem that there should not here be any insuperable obstacle to common ground discussion, even though the points of view are somewhat different.

If I understand Jung's position he places emphasis on the episode of the moment and pictures the libido as flying back to some psychic or physical episode of earlier life, but not of so early date as that which Freud designates richest in polymorphous sexual material.

It is in relation to this subject that Jung uses the expression "*Die Libido . . . wurde nicht anerkannt*," thereby causing Abraham much indignation, who says, "*Jung scheint den Terminus 'Verdrängung' gefliessentlich zu vermeiden*." Let us suppose that for some reason Jung does object to and side-step the term *Verdrängung*. If he does prefer the expression "*die Libido wurde nicht anerkannt*," is there of necessity any denial of the psychic mechanism so fundamental to the teaching of the Vienna school? Does not the lack of *Anerkennung* of libido postulate conflict, which in turn postulates an inhibiting psychic force? And if this be so is not the difference more one of terminology and not psychological concept?

Another point which does not in the least affect the main issue is Jung's disinclination to accept the infantile sexual phenomena as perversities. He says, "*Der frühere kindliche Zustand aber ist eine normale Anwendungsweise der libido, während das zurückkehren der Libido auf die infantilen Wege etwas abnormes ist. Ich bin daher der Ansicht, dass Freud nicht berichtigt sei, die infantilen sexual Erscheinungen als pervers zu bezeichnen, indem eine normale Erscheinung nicht mit Terminis der Pathologie bezeichnet werden darf.*"

This is of academic interest only, and can be freely admitted or contested, and still no change in anything but definition involved.

And right here it were well to quote from Schiller's work on "Humanism," where he deals with definitions:

"The difficulty, moreover, of defining adequately is indefinitely increased when we have to deal with subjects of which our knowledge, or their nature, is rapidly developing, so that our definitions grow obsolete almost as fast as they are made. . . . And so it is the duty of those who labor at such subjects to avail themselves of every opportunity of explaining what they mean, to begin with, and never to weary of redefining their conceptions when the growth of knowledge has enlarged them, even though they may be aware that however assiduously they perform this duty, they will not escape misconception, nor, probably, misrepresentation."

Now, after all the turmoil of wordy conflict is over, what will still remain to the Vienna and Zürick schools in common?

In answering this question we must use definitive terms, and thereby at once throw ourselves open to opposition from the truculent. But let him who objects to this term or to that, substitute his own. He may change a term, but cannot change truth. The psychological fact of polymorphic sexual content in the infant is just as vital a truth if called by one name as by another. Our interest centers on the fact being realized.

With this understanding we can safely affirm that there is common point of view, with a healthy quantitative difference of opinion, in infantile polymorphic sexual content, in fixation, in regression. Use what terminology you will, the mechanisms of *Verdrängung*, censor action, conflict, resistance, compromise, conversion and transference are still acknowledged as fundamentally correct, though the emphasis in certain questions may vary with different investigators.

The interpretation of dreams, word-association and free-association, are means to an end. They will, it is to be hoped, be subject to a healthy evolution and, like surgical instruments, be constantly improved, which means changed.

In his concluding paragraph Jung says: "*Es war mein Bestreben, gewisse von den Freudschen Hypothesen abweichende Ansichten nicht als kontradiktorische Behauptungen aufzustellen, sondern als organische Weiterentwicklung der von Freud in die Wissenschaft eingeführten Grundgedanken darzustellen.*"

I do not see how one can take issue with this spirit. I do not see how a quiet, orderly controversy between Jung and Freud, on disputed points, can do otherwise than advance our knowledge of psychopathology. I do not see that anything Jung has said calls for the savage and contumelious criticism meted out by Abraham or Furtmüller.

We in this association represent many divergent points of view. This in itself means potential for tremendous development if we take advantage of it as scientists with a common interest should. But when it comes to bigoted acrimony and the striving to overwhelm by brute force rather than reason, let us leave that sorry picture to the church in the middle-ages where it rightly belongs.

247

Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 41, No. 7, July, 1914.]

PARAMYOCLONUS MULTIPLEX, INCLUDING A CASE WITH NECROPSY SHOWING LYMPHOCYTIC INFILTRATION OF THE PIA[1]

By EDWARD MERCUR WILLIAMS, M.D.

SIOUX CITY, IOWA

FORMERLY INSTRUCTOR IN NEUROLOGY AND NEUROPATHOLOGY AT THE UNIVERSITY OF PENNSYLVANIA AND ASSISTANT NEUROLOGIST TO THE UNIVERSITY HOSPITAL

So few cases of paramyoclonus multiplex with necropsies have been reported and of these only one with lymphocytic infiltration that I thought another of this type would be worth recording. In addition to the necropsy findings, some of the clinical features were unusual, such as the involvement of the face and the tongue, the increase of the movements during voluntary efforts and the peculiar affection of the speech. For the history and material of this case I am indebted to Dr. W. G. Spiller. The others are selected from the dispensary service of the University of Pennsylvania, the East Oklahoma Hospital for the Insane and my private work.

Paramyoclonus multiplex was described by Friedreich in 1881 as a symptom complex in which occurred quick, jerky contractions of the muscles. The distribution of the contractions was generally symmetrical and included principally the trunkal muscles and those least under voluntary control. The movements were confined to bundles of fibers or individual muscles, resembling fibrillary or fascicular twitchings, or causing a bulging of the entire muscle and occurring at the rate of thirty to one hundred and twenty per minute. The joints and limbs were, as a rule, not moved by these jerkings, or, if so, only slightly. The muscles of the face were not usually affected, the platysma, however, was one of those most frequently involved. The contractions ceased when the attention was directed to them, due to the psychic control, but became worse when the mind was diverted, as when the patient would lie down and think of something else. Marie, Bechterew and Stadler referred to the importance of this. The symptoms disappeared entirely during sleep. In one of my cases, however, the attacks always came on when the patient appeared to be sleeping profoundly.

---

[1] Read at the fortieth annual meeting of the American Neurological Association at Albany, N. Y., May 7, 8 and 9, 1914. From the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania and the East Oklahoma Hospital for the Insane.

Since the appearance of Friedreich's original article numbers of cases have been described under this name. Owing, however, to the great similarity in the character of the movements occurring in other more or less allied conditions the definite understanding of paramyoclonus as a clinical entity is rather obscure.

The cases conforming to Friedreich's original description, where some psychic disturbance is the evident exciting cause, often border so closely upon hysteria that it is difficult to see why they should not be considered as myoclonic or myoclonoid manifestations of hysteria rather than as paramyoclonus multiplex.

Lukacs, Verzar, Sizaret, Bernard and a number of others recognized the importance of trauma and mental shock in the origin of the disease and in Friedreich's case a fright preceded the onset of the affection.

Strümpell, Houchard and Fleisinger regard paramyoclonus multiplex as identical with hysteria, while Hoffmann and Boettinger refer to its close relationship to, or identity with, chronic chorea.

Farge, Heilig and Mettler believe most cases to be only symptomatic or transitional.

Unverricht has considered quite exhaustively a familiar form of myoclonia occurring with epilepsy in the degenerative type of neuroses. Lunborg, Bührer, Garnier, Verco, Putnam, Mott and others have also recorded cases of this character. In these reports motor symptoms were the same as in the functional type of paramyoclonus, with the addition of true epileptic seizures.

In these epileptic forms, according to Unverricht, the tongue and throat are most frequently involved, whereas affection of the face from the onset of the disease, in Heilig's opinion, indicates a form of hysteria, tic, or chorea.

Reynold reports the occurrence of myoclonic movements between the true epileptic seizures in about 75 per cent. of all epileptics. Ballet, Bruns, Hoffmann, Reynolds and others also refer to the presence of these interparoxysmal myoclonias. Such patients for a long period may have the true epileptic seizures only during the night when they are not observed. The question of making a definite diagnosis and prognosis in some of these doubtful cases is both a serious and a difficult one. One might suppose himself to be dealing with a purely functional condition and later have the patient develop an active and unmistakable epilepsy. Instances of this kind have been reported (Turner) and such a transition was shown in one of my cases. To make the problem more difficult hysteria doubtless occurs in conjunction with both the functional and the epileptic forms. Ziehen, Venturi, Marina De Concilius and Carriere mention this relation and it was found in case five of this paper. I have found these combined forms wrongly called hystero-epilepsy, a term so often misused by the general profession.

From the histories of seven hundred and five epileptics in the dispensary of the University of Pennsylvania, many of whom I questioned particularly about the possible occurrence of interparoxysmal movements, and from the study of eighty-nine epileptics, whom I observed for a number of months at the East Oklahoma Hospital for the Insane, I have been unable to confirm Reynolds' view. Myoclonic movements were present between the epileptic seizures in only three of the dispensary cases and in three of the patients at the hospital. The histories of these patients are of interest and are given in a brief way, no mention being made of tendon reflexes, Babinski signs, etc., where they were normal.

Shanahan mentions the occurrence of paramyoclonus in only 7 of the 2,150 cases of epilepsy he observed and Alden Turner refers to only 2 cases among the 2,000 epileptics reported in his work. Singularly in both of these the paramyoclonus antedated by several years the genuine epileptic seizures.

Soury believes paramyoclonus multiplex to be similar to catalepsy and the hypnotic state, in that the psychic control is gone and the muscles, instead of retaining their normal responsive power and position, react to stimuli by irregular, jerky and incoordinated movements.

As already mentioned, paramyoclonus occurs symptomatically with, or following various disorders and is so reported by many writers. Indeed, I do not see that even the clearest cases deserve recording as true clinical entities, but believe the attacks should be considered as myoclonic, or myoclonoid manifestations of hysteria, epilepsy, or whatever the underlying disease might be. Frequently the resemblance to the tics and choreas is so close that the attacks might be forms of these affections.

Paramyoclonic movements have been observed following diphtheria (Remak) and other infectious diseases (Raymond, Valobra, Sterling, Meynier). As a complication of lead poisoning Leubuscher mentions them and Starr, Carriere, Bertrand and many others report them following trauma. They also occur in paresis (Grawitz) and in a case of paresis at the East Oklahoma Hospital for the Insane, muscular twitchings occurring at the rate of about 100–120 per minute were present for a period of two days. They included the facial muscles on one side (eyelid, forehead, mouth) in such a way as to give the expression of unilateral laughing. These intermittent twitchings were accompanied for an hour or so at a time with peculiar spasmodic grunting sounds resembling low laughter. These apparently emotional outbreaks would last one or two, sometimes five, seconds and would again occur after an interval of about a half minute.

Clark described myoclonic movements in meningitis and encephalitis and I have seen them in a case of encephalitis following influenza.[2]

Stadler refers to the resemblance to Thomsen's disease. Friedreich believed the condition was due to irritation of the anterior horn cells. Tutschaninow had a similar view and seemed to verify it by experimentally producing spasms in dogs with spinal injections of carbolic acid.

Popow claimed a myopathic origin while Homer and Vaulair believe the cause to be a hypersensitiveness of the receptor cells of the cord. A very interesting case reported by Strasman with the changes in the receptor side of the cord typical of tabes, such as lost tendon reflexes, diminished sensation, etc., contradicts this view of Homer and Vaulair.

---

[2] In discussing one of the papers at this meeting Dr. Pierce Bailey referred to a case of paramyoclonus occurring with anterior poliomyelitis.

Lunborg thought intestinal intoxication very important as a provocative cause in the epileptic forms.

Necropsies have only added to the obscurity of the etiology of paramyoclonus. Cortical irritation was found as a cause, particularly where the attacks were of an intermittent or symptomatic type (Patella, Pairot, Pic and others). Hypertrophy of the muscles was found by Hunt, whereas Stadler reports muscular atrophy in two cases.

CASE I. R. C. Colored laborer, age 62. Admitted to the Philadelphia General Hospital in March, 1909, complaining of "Nervousness and shaking all over the body."

The family history was negative. Patient used alcohol moderately, but used a great deal of tobacco.

Examination.—Well-nourished man, apparently about sixty-five years old. No scars on his body. Skin dry, harsh, and scaly. Nearly every muscle of his body showed coarse tremors or twitchings and his voice was tremulous and stuttering, making it very difficult to understand him.

His gait was very slow; he took very slow, shuffling steps, raising his feet only slightly from the floor. Attempts to turn quickly caused ataxia and a marked coarse tremor of the legs and arms. On standing he swayed slightly. This was not increased by closing his eyes.

Every attempt on the patient's part to move voluntarily any part of the body provoked a coarse tremor. Thus, wrinkling the forehead caused trembling of the eyelids and occipito-frontalis and with the attempts to draw up either corner of the mouth there were tremors of the cheek, mouth and neck muscles. The moment the patient relaxed the tremors ceased. The tongue was large, flabby and coated, possibly very slightly atrophied on the left side and a very violent coarse tremor was noted when the tongue was protruded, freely movable in all directions. With the exception of the above motor disturbances, the cranial nerves were normal. Posterior cervical glands were not enlarged. Irides reacted normally to light and accommodation and pupils were round and equal.

The musculature of the arms was fair, no atrophy nor spasticity; active and passive movements normal as to power and resistance, etc. Biceps and triceps reflexes prompt and exaggerated on either side and equally so.

Deformity of left wrist from an old fracture. Grip weak on that side; stronger on the right, but weaker than it should be.

Upon voluntary efforts the muscles of the hands and arms developed coarse tremors, subsiding in a few moments after the discontinuance of the exertion. When at rest and supported there were no tremors. There were, however, constant fibrillary twitchings of the right biceps during the observation, increasing and decreasing at times. Fibrillary twitchings of a finer type were also noticed in the upper part of the left pectoralis major.

Hands extended showed coarse tremor, more intense on the right.

The muscles of the legs were weak, not atrophied nor spastic. Could perform all movements. Achilles and patella tendon reflexes normal. No clonus. No Babinski. Senation tested for heat, cold, pain and touch, was found to be normal. Bladder and rectum not affected. Attempts to sit up caused a coarse tremor of the entire body.

Finger to nose and finger to finger tests showed slight ataxia. The ataxia was much greater in the legs largely due probably to the greater comparative weakness.

Mental power was fair. The speech was stuttering, tremulous and very jerky. The lips and tongue trembled when the patient tried to talk to such an extent as to make it almost impossible to understand him.

Patient contracted pneumonia and died three weeks after admission.

The brain and cord were removed at the necropsy and later sections were taken from the usual areas of each. As the findings did not show any difference except in intensity in the portions examined, only several representative areas will be included and they are as follows: left paracentral convolution, the pons, the cervical, the thoracic and the lumbar segments of the spinal cord.

Paracentral Convolution.—The meninges were infiltrated with round cells, extending into the cortex as an intense perivascular infiltration. Plasma cells were present, but not very numerous. The vessels were congested. Numerous small vacuoles were found throughout the sections, but they were not perivascular and may have been artifacts or due to bacilli (?). Under high magnification and careful focusing there appeared to be a groundwork of lightly staining tissue at a different plane from the remaining portion of the section, so that these cavities may have been simply due to some unusual contracted condition occurring in portions of the section more thinly cut than others, either due to a poor knife, or due to cutting over a space occupied by a shrunken ganglion cell, or one destroyed by neuronophagocytosis.

The cells stained poorly and many of the nuclei were absent or not centrally placed. Neuronophagocytosis could be found in various stages. The glial cells were somewhat increased.

Pons.—The meninges were infiltrated with lymphocytes, these extending into the substance of the pons. Vessels of the pia and the intra-pontine vessels were congested. In many places they contained what appeared to be hyaline clots. The coats of the vessels were thickened. A peripheral zone of degeneration and many areas of perivascular rarefaction of tissue were present. The cells showed poor staining, probably due to the age of the specimen. No tract degeneration.

Cervical Cord.—The posterior roots were degenerated and atrophied and the interstitial tissue increased. The thickened pia showed extensive extravasation of red cells in the vessel coats and in the surrounding tissue. The cells were in various stages of chromatolysis. The pigment was increased, nuclei destroyed, or displaced, so that only perhaps one tenth of the cells approached the normal.

Thoracic Segments.—Pia thickened and infiltrated and its vessels intensely congested. Amyloid bodies here as in cervical areas were numerous and the zonal degeneration was well marked. There were no tract degenerations. The anterior horn cells were reduced in number and those remaining were in various stages of dissolution, practically none of them being normal.

Lumbar Segment.—The anterior and posterior roots were degenerated, giving to many of the fibers a constricted beaded appearance. Amyloid bodies were present within the posterior root bundles and in the substance of the cord. The pia was somewhat thickened, slightly infiltrated and adherent to the cord. The vessels were thickened and congested. Considerable extravasation of erythrocytes in the pia. The ganglion cells showed different degrees of degeneration, the greater part of them closely approximating the normal.

CASE 2. J. B., No. 7446. Male, age 24. Came to the dispensary of the University of Pennsylvania. Had attacks of twitching of the muscles of the body and jumping of the legs. When this occurred the patient knew he was going to have a seizure and in about an hour he would become unconscious and fall. Claimed that he remained unconscious fifteen to twenty minutes. Had never injured himself during an attack.

CASE 3. A. B., No. 7048. Girl, age 6. Came to University dispensary in 1911. History of a normal birth, sat up at six months and walked at age of one year. Head was bumped against a gas fixture at this age and shortly after she began to have attacks of unconsciousness about twice a month. During these seizures had the typical signs of epilepsy. Also had attacks of sudden jerking forward of the head without loss of consciousness. These movements have been so violent as to break her nose and cut her face and head.

CASE 4. O. G, No. 5633. Male, age 19. At the University dispensary. Maternal grandmother had epilepsy. Patient healthy up to age of fifteen, when he was struck upon the head with a window sash. A year later had a spell of unconsciousness (doubtful). A year after this began to have attacks of jerking of the hands and legs without loss of consciousness. These attacks occurred at intervals of about a month up to the fifth month, when he had an attack of this character, lasting the entire day, but without loss of consciousness, and after supper the same day had a typical epileptic seizure with loss of consciousness, frothing at the mouth, etc. Since then he has had only the typically epileptic seizures.

CASE 5. T. T., woman, age 32. Inmate of the East Oklahoma Hospital for the Insane. Had typical epileptic seizures since childhood. Several relatives epileptic. In addition to the regular seizures she had attacks of twitching and jerking of all the muscles of the body including neck, but only occasionally the face. The movements are bilateral, symmetrical and produce at times a violent trembling. The patient gradually sinks to the ground during these attacks, but never injures herself. She does not lose consciousness and the attacks could be brought on by suggestion, or even by pointing the finger at her. She had the mean, irritable disposition of the epileptic and had frequently bitten other inmates with practically no provocation. Her epileptic seizures were unmistakable, as they had all the symptoms of true major epilepsy. They occurred rather irregularly, being influenced more or less by her course of treatment.

CASE 6. Mrs. A. W., age 55. Inmate of the East Oklahoma Hospital for the Insane. History of epileptic seizures and attacks of shaking as long as patient could remember. Several members of the family epileptic. Her ordinary seizures were unmistakably epileptic. Between these seizures and occurring without any relative time to the regular attacks, that is, not being in the sense of an aura, etc., she had spells of shaking and jerking of almost all the muscles of her body. She would feel this jerking and be conscious throughout. Under bromide treatment she had only an occasional seizure of the epileptic type.

CASE 7. W. H. Male, age 25. Inmate of the East Oklahoma Hospital for the Insane. Low-grade epileptic imbecile. No epilepsy in family (?). Very severe attacks of epilepsy since childhood. Practically unaffected by treatment. At times had generalized jerking movements without loss of consciousness. These attacks were reduced in frequency by the bromide treatment.

CASE 8. Mrs. B., age 28. For a number of years at different periods has had "spells" of a peculiar kind. Often they would come on during the night, apparently during a sound sleep, and the patient would become cyanosed, once or twice frothed at the mouth and bit her lips. She is or was always sleepy after the attacks. Has voided once or twice during the seizures.

At other times the patient has attacks of trembling, that is, she feels as though she is trembling, but in reality only the muscles of the legs and abdomen are twitching, and she does not shake or move.

Sometimes she would have so-called nervous chills. During some of her spells the patient was unconscious, though in most of them she knew what was going on, but seemed dazed. Never falls nor hurts herself, as she is always aware of the onset. They are brought on more often by some excitement, such as a family disagreement, etc.

The series of attacks will extend over a period of a couple of weeks, then clear up for an indefinite period. She is usually thin and nervous after them.

The present outbreak came on following an evening at the moving picture show, where one of the characters was probably an epileptic, and had rather peculiar, atypical seizures. In her attacks now—they were formerly general—the left arm, neck and face are mostly involved. She cannot swallow during a spell.

In one of the observed attacks the left side of the face twitched about the rate of 2—4 to the second—the face flushed and tears came to the eyes.

A couple of nights later she had a severe spell in which she drew up her legs and arms, but there were none of the rapid jerky movements. Appeared to be confused and irritable and did not want to remain still—was emotional, muttered and cried. Did not want to talk after the attack, and when she did it seemed as though her tongue were affected in some way. Slight incontinence of urine in one of the spells during the day. Knee jerks increased —no Babinski—has voluntary extension of all the toes. No sensory changes nor paralyses. Eye grounds normal. Reflexes normal during and after the spells.

A very close resemblance could be traced to the seizures of the actor in the moving picture play.

LITERATURE

De Concilius. Ann. di med. n. Roma., 1910.
Friedreich. Virch. Arch., 1881, Bd. 86.
Garnier. Ann. méd. psych., Paris, 1898.
Heilig. Arch. f. Psych. u. Nervenk., 1911.
Lunborg. Die Progressive Myoklon Epilepsie, Upsala, 1903.
Lunborg. Neur. Centr., 1904.
Mettler. Diseases of the Nervous System, 1905.
Pic. Nov. Icon. d. Salpêt., 1904.
Putnam. Buffalo Med. Jour., November, 1911.
Reynolds. Cited by Soury.
Shanahan. Jour. Nerv. and Ment. Dis., August, 1907.
Sizaret. Ann. méd. psychol., Paris, 1909.
Soury. Ann. méd. psychol., Paris, 1897.
Strasmann. Ztschr. f. d. gesamm. Neur. u. Psychol. Original., Vol. X, 1910.
Turner, W. A. Epilepsy, 1907.
Unverricht. Die Myoklonie, 1891.
Unverricht. Deut. Ztschr. f. Nervhk., 1895.
Verco. Australasia M. Gaz., Sydney, 1912.
Other authors cited by Heilig.

2.59

Experience with Crotalin at the Oakbourne Epileptic Colony

N. S. YAWGER, M.D.
PHILADELPHIA

EXPERIENCE WITH CROTALIN AT THE OAKBOURNE EPILEPTIC COLONY *

N. S. YAWGER, M.D.
PHILADELPHIA

The treatment of epilepsy by the injection of rattlesnake venom has attracted some attention. Those of us who are connected with the Pennsylvania Epileptic Hospital and Colony Farm have been besieged by inquiries as to its value. Information was sought in person and by telephone, and letters were received from different parts of the country. The glowing reports heralded throughout the land in the lay press reached our patients and their families, some of whom almost insisted on our using crotalin. The matter became somewhat troublesome and, since we were without personal knowledge on the subject, there seemed sufficient reason for our giving the venom a trial.

Observations on epileptics made under colony supervision should be useful, since the life there is the most regular and healthful possible. Patients are not left to report on their own condition—a method of gathering information which may be most unreliable. Attacks often occur while patients are asleep, and frequently there are manifestations during the day of which these persons are unaware. Again, seizures may prevent them from reporting regularly to the physician. These conditions and others detract from the value of the work when the patients are not kept under close observation.

For the purpose of this study a fair group of patients having idiopathic epilepsy was selected for three months' treatment. Afterward came the work of leveling up to determine as nearly as possible just

---

* Read before the Philadelphia County Medical Society, April 15, 1914.

* From the Pennsylvania Epileptic Hospital and Colony Farm and the Department of Neurology of the University of Pennsylvania.

what effect crotalin had on these patients. This was a difficult problem, and a few words parenthetically may not be out of place.

Epilepsy is perhaps the most peculiar disease known. Aside from their epileptic phenomena and characteristic temperament, some of these patients are in moderately good physical condition. All paroxysms may cease for a time without known cause and in very rare instances a spontaneous cure has occurred. An acute illness usually holds the seizures in abeyance, and pregnancy occasionally does the same. Some epileptics have attacks only when asleep, and there are a few whose disease is never suspected. Seizures may be so mild as scarcely to be observed even while the patient is watched, or status epilepticus may supervene at any time, and this condition is often fatal by the third attack. About one out of ten epileptics has frequent insane episodes which occur precipitately, and there is no more dangerous form of insanity. Self-consciousness may be lost suddenly, and in this state of automatism the worst of crimes have been committed without the subsequent knowledge of the patient. When in such a condition the subject is no more responsible for his actions than we are for our dreams.

There is a great deal more to epilepsy than fits; indeed, the cardinal symptom of epilepsy is the disturbance of consciousness and not the motor manifestation.

In a series of cases no course of treatment has been highly gratifying, and sometimes disappointment comes when it is least expected. A patient now under observation is a case in point. She is a 14-year-old schoolgirl at our colony, and was having on an average twenty-five seizures a month. Under active thyroid treatment she made decided improvement in every respect, and for six months was without attacks. Of course we thought we were on the right track. And then, without known cause, she began with a few isolated attacks and a little later suddenly broke into a series of 142 seizures which were by far the worst she has ever experienced.

Our observations were made on six patients who had idiopathic epilepsy. All but one were given an intramuscular injection of crotalin once a week for three months. We began with $^{1}/_{200}$ grain, and the amount was gradually increased to the strength mentioned in each case.

A distinct psychic influence is often observed in the treatment of epileptics, and in view of this the purpose of the injections was kept as much as possible from the knowledge of the patient.

Case 1.—A. P., a Hebrew girl aged 16, had been a resident of the colony for two years. Her attacks began at 8 years of age without obvious cause. She was one of the best pupils in our school, but still was just a shade below the average girl in mentality. The seizures were both major and minor. She had never had status epilepticus, nor had there ever been an insane episode. When treatment was begun her attacks were about as frequent as at the time of admission. The injections were gradually increased to $^{1}/_{50}$ grain. This girl always had an irritable disposition; she improved somewhat in this respect. For the year previous to treatment she had an average of eighteen attacks a month and during treatment there was an average of seventy-nine seizures a month.

Case 2.—J. C., a boy aged 16, had been a resident of the colony for four and a half years. His attacks began in infancy without known cause. The clinical history of this patient showed two attacks of status epilepticus which were followed by stupor for about four weeks. He was feebleminded. For the year previous to treatment there had been an average of twenty seizures per month. After treatment for a month his attacks began to increase in number, and then status obtruded itself and this was followed by a stuporous state which persisted throughout the treatment. The final injection was $^{1}/_{40}$ grain. The status was somewhat confusing but so far as we could determine, this patient was uninfluenced by treatment.

Case 3.—L. M., a woman aged 34, had been a resident of the colony for three years. Her attacks had begun fifteen years ago and were about as frequent as at the time of admission. She was feeble-minded. The final injection was $^{1}/_{30}$ grain. For the year previous to treatment her attacks occurred twelve times a month and during treatment there were about thirteen a month. This patient was apparently uninfluenced by treatment.

Case 4.—E. M., a woman aged 32, single, had been a resident of the colony for nine years. The first attack occurred at 19 years of age just after the death of her mother. The attacks were both major and minor and at the time of admission were occurring about five times a day. Occasionally the seizures were in series. The intelligence of this patient is that of the average epileptic. She has always showed some mental depression and has suffered frequently from headache. At one time she was placed for three months on a drug prominent in the armamentarium of the epileptologist, and made very decided temporary improvement. During the last month of this period she had only four very light attacks; she was mentally alert and showed less depression than at any time during her residence at the colony. Unfortunately she, like so many others, relapsed. It was thought that this patient might prove responsive temporarily to crotalin also, but an unusual condition developed. After the first injection her arm became intensely swollen, much more so than those of the other patients. For the week following she had twenty attacks. The second injection, which was 1/150 grain, gave rise to more marked local reaction and for the ensuing week there were fifteen attacks. The third injection was 1/125 grain, and the local disturbance resulting was most intense. During the following week there were twenty-two seizures and these were more severe than any that had occurred during the patient's nine years' residence in the colony. In addition to the cellulitis there was prostration, extreme mental depression, loss of appetite and nausea; she had constant and severe headache. Because of these symptoms the injection for the following week was omitted, when her condition promptly improved and she had only twelve minor attacks. A week later, 1/100 grain was given, and ten attacks were recorded with a return of all the toxic symptoms. Another injection was tried the next week, with the result that fifteen severe convulsions followed and the toxic symptoms which ensued were so severe as to cause us to feel that further trial was unjustified.

Case 5.—C. A., a man aged 35, a clerk, had been a resident of the colony for seven years and was usefully employed there. He was married at 27 years of age and his first seizure occurred four months later. Epilepsy developing at that age is sometimes on a syphilitic basis. The ophthalmologic examination by Dr. H. Maxwell Langdon, the Wassermann test by Dr. Corson-White and the absence of luetic neurologic findings all spoke against syphilis. Some years before there had been an insane episode of two days' duration. This patient's mental condition was better than that of any other of the adult colonists. He was considered a good subject for treatment because of his preserved mentality, and beside he was exceedingly desirous and we may say almost insistent in treatment. He had come to the city and consulted a physician and would have left us to take the crotalin treatment had he had the money to do so. Attacks for the year previous to treatment were about as frequent as at the time of admission and amounted to three severe seizures a month occurring in series. The final injection was 1/25 grain. During the treatment he had an average of five attacks a month, he was exceedingly irritable and quarrelsome and his memory was somewhat impaired. He realized that he was worse than before and asked that the treatment be discontinued.

Case 6.—The family of this patient was exceedingly anxious that the treatment be given. J. C., a man aged 23, single, had been a resident of the colony for one year and three months. His attacks began at 9 years of age without known cause. His clinical history showed that he had had a few brief periods of mental aberration. The last injection given was 1/25 grain. Previous to treatment there were on an average of thirteen attacks a month, and during treatment there were twelve attacks a month; but in the midst of the course he had an insane period of three weeks' duration during which time he was free from seizures. His subsequent history included three insane periods of short duration and three series of major epileptic attacks. The last series occurred two and a half months after treatment had stopped. During the last series, which was of two days' duration, sixty convulsions occurred and then he died.

Briefly stated, our experience with crotalin in the treatment of six cases of idiopathic epilepsy was this: Two patients were uninfluenced; two were worse during the treatment; one, early in the course developed such intolerant toxic symptoms that further experimentation was unjustified, and the last patient died two and a half months after treatment. While we did not feel that death resulted from the use of crotalin, the patient's disease certainly was not benefited by the treatment.

2005 Chestnut Street.

Reprinted from The Journal of the American Medical Association
May 16, 1914, Vol. LXII, pp. 1533-1535

Copyright, 1914
American Medical Association, 535 N. Dearborn St., Chicago

Extracted from the American Journal of the Medical Sciences,
May, 1914, No. 5, vol. cxlvii, p. 735.

ALCOHOLISM AND EPILEPSY, ALSO SO-CALLED ACUTE ALCOHOLIC EPILEPSY.[1]

By N. S. Yawger, M.D.,
EXAMINING PHYSICIAN TO THE PENNSYLVANIA EPILEPTIC HOSPITAL AND COLONY FARM,
PHILADELPHIA.

(From the Department of Neurology in the University of Pennsylvania.)

The craving for alcohol and alkaloids or substances that are similar to these in their action is so universal as to appear almost instinctive. From cereals, fruits, plants, seeds, and tubers, man has obtained substances for such use, and those commonly employed have been the various alcoholic beverages, cocoa, coffee, guarana, hemp, kola bean, maté, opium, tea, and tobacco. Such substances have been drunk, chewed, smoked, inhaled, used subcutaneously, and otherwise introduced locally. Habits of this kind were in vogue among the ancients even in the earliest times. There are records that tell of the use of wine 5000 B.C. And since it appears that man always has craved something to soften his sorrows and to add to his pleasures, there is no likelihood that we shall become absolutely free from this human weakness.

Having, then, these habits always with us, it is of the utmost importance to be familiar with the harm that results from such indulgence. This is a difficult problem, for upon no subject within the domain of medicine do such extreme views obtain as upon that of the use of intoxicants. Of all habit-engendering substances, alcohol is the one most commonly used, but a number of factors must be borne in mind in determining the effects produced by such use. The quantity and the quality of the liquor; the time of day taken, and the period of time over which it is consumed; and not the least important factor is the physical and mental make-up of the individual. The matter of tolerance is significant. It has been defined by Hare, of England, "as the capacity to ingest relatively large amounts of alcohol without experiencing the ordinary obtrusive physiological effects."

---

[1] Read before the Chester County Medical Society at Embreeville, Pa., July 8, 1913.

Although we know that at times alcohol may be used in enormous quantities and for a long period without showing appreciable deleterious effects, we also know that it may cause death acutely or it may bring about widespread degeneration through years of excessive indulgence. The pathological changes extend to all tissues except bone, but its effects are especially marked in the nervous system. The particular affinity of alcohol for this part of the body is shown even after death, when it may be found in the cerebrospinal fluid after all trace has disappeared elsewhere. In the nervous system both functional and organic disturbances are observed. Mental manifestations with alcoholism as a factor are especially numerous, and any type of insanity may be simulated. However, the only mental disorders which are distinctly the result of the prolonged action of alcohol upon a previously normal brain are delirium tremens and polyneuritic psychosis (Korsakow's disease), and even the latter is occasionally produced by the absorption of septic matter, and it is also said to have followed influenza and typhoid. All other psychoses occurring among alcoholics are of the types of those developing among abstainers.

Many believe that in the offspring of alcoholists there is a proneness to hysteria, insanity, idiocy, epilepsy, and crime. But here it is important to remember that the nervous instability which may have led the parents to drink, may also account, at least in part, for these disorders and diseases developing in the offspring.

At times information is gathered that is worse than useless; it is misleading. Mott, a distinguished English writer, says: "The great point in any scientific observation is not to try to prove something, and to avoid any propagandistic tendency, thus the question of alcohol and insanity is an illustration in point. The Council of Fifty in Massachusetts investigated the number of patients admitted to asylums in which there was an alcoholic history; it was then suggested that they might investigate the number of total abstainers; it was found that they were quite as numerous."

Of special interest is the relation of alcoholism to epilepsy; it is concerned with this disease in several ways, and yet it probably is not as potently a factor in the causation of epilepsy as is often stated, for we have not sufficiently taken into consideration the nervous instability which frequently has led the parents to drink, and which in itself must have been partly, and at times perhaps wholly, responsible for the development of epilepsy in the offspring. It also appears that only rarely does chronic alcoholism produce recurrent attacks of fits, except as they are dependent upon the immediate taking of alcohol. This latter subject will be considered somewhat in detail later.

Alcoholism has, or is alleged to have, the following relations to epilepsy:

1. Drunkenness of one or both parents at the time of conception has been said to be a fruitful cause of epilepsy in the offspring (Esquirol). This is an exceedingly difficult matter to investigate, as the nature of the sexual act forbids observation. However, there is data which indirectly throws light upon this subject. In France it is said that a considerable number of children appear nine months after seasons of drinking, such as carnivals in the cities and church fairs in the country. It has also been observed in some countries that the congenital epileptics and imbeciles are conceived at the time of new wine. It is held, as a rule, in the wine countries that a poor school year comes seven years after a good wine year.

2. That injuries to the head in alcoholists may give rise to true epilepsy is stated by Oppenheim. This is quite possible just as alcoholism plus traumatism may cause insanity; and also, a blow upon the head in a syphilitic may act in the same way.

3. Chronic alcoholism in the parents may lead to epilepsy in the offspring; while reliable statistics are here wanting, it is probable that this is often one of the factors in the production of epilepsy.

4. Chronic alcoholism in the individual may lead rarely to epilepsy in those with no inherited or inherent tendency to the disease. This surely does not often follow except in cases of senile epilepsy, where the attacks are rather an epileptoid manifestation and dependent upon an arteriosclerotic basis. Such attacks are less frequent and less severe, and the mental failure is not so marked, the condition also yields somewhat more satisfactorily to treatment than does the idiopathic disease.

5. In an epileptic who becomes an alcoholist, the attacks are increased in number and in severity. Of the marked intolerance of epileptics to alcohol, there can be no question. All epileptologists have met with instances where the disease has undergone a remission and where attacks have been again precipitated by the taking of alcoholic beverages.

6. Alcoholic drinks may be sought by epileptics because of post-epileptic depression, and there may be little or no drinking at other times. The disease, of course, is exaggerated.

7. A few epileptics upon becoming aware of the dreadful malady with which they are afflicted, have plunged into drink and greatly aggravated their disease; and this, independent of the depression which is sometimes the direct result of the attacks. Here, epilepsy is the exciting cause of alcoholism.

By those in charge of alcoholic wards in hospitals, the phenomenon of convulsions is frequently observed. This may be due to uremia, and it also occurs in epileptics who have gotten drunk. But there is another variety of convulsive disorder seen, and this has received but little attention at the hands of writers upon alcoholism; some even fail to mention it. We refer to so-called acute alcoholic epilepsy. The term "epilepsy" is here unfortunately used, since these attacks have no more relation to epilepsy than have the convulsions so commonly seen in children. As a rule, there is but little albumin in the urine of these patients, and this usually clears up in a few days. Contrary to the belief of a number of physicians, alcoholic convulsions and delirium tremens appear in most instances to be abstinence phenomena. They do not usually occur together, although occasionally this is observed. It is argued that these two diseases sometimes develop while the patient is still drinking heavily; in some of such cases perhaps there has suddenly developed a catarrhal condition which has prevented the absorption of alcohol; or it may also arise through a patient being profoundly intoxicated and therefore not conscious enough to continue drinking. These fits are most apt to occur in continuous alcoholists in whom a rather high degree of tolerance has been attained, and they are most likely to obtrude themselves during the first twenty-four hours of abstinence. When attacks of delirium tremens are present they occur a little later. These convulsions are not preceded by insomnia, tremor, and fear unless they are accompanied by delirium tremens. The attacks bear considerable resemblance to genuine epileptic seizures. They are mostly major; an injury may be sustained by falling, unconsciousness is complete, the tongue at times is bitten, sleep often follows the attack, and the patient usually awakens with a headache. But still there is not the characteristic appearance of an epileptic, and the patient usually bears the stamp of a chronic alcoholic; the attacks seldom occur independently of drinking.

The prominence given alcohol as a cause of epilepsy has been overestimated, and this is apparent from the following: The nervous instability which at times drives parents to drink is in part responsible for the development of epilepsy in the offspring. We know that drinking is very common and epilepsy is relatively rare. Alcoholism is much more frequently encountered in males, but epilepsy at least is as common among females. Many of the cases of epilepsy have developed before the age when drink could be obtained. Insanity and nervous diseases are common among the Jews; epilepsy is frequently seen, and yet the Jews are an abstemious people. Those of us who have done special work among alcoholics know that a drunken Jew is a great rarity. The report of the Craig Colony for Epileptics for 1912 gives alcoholism as the probable cause of epilepsy in less than 1 per cent. of the admissions for that year, 227 patients were admitted. And, finally, even those alcoholists with no inherited or inherent tendency to epilepsy, who have convulsions (so-called alcoholic epilepsy), almost never have fits independent of drinking.

A great deal is heard about the rapid increase in the number of epileptics, but the writer believes here we stumble against some of the errors which have arisen in estimating the number of insane. It is only in recent years that we have had special institutions for the care of epileptics so that we are only just beginning to obtain data from which to draw more definite conclusions as to etiology. The conditions conducive to epilepsy and insanity may not parallel each other, but they probably do run in much the same direction.

The writer knows of no reason for believing that through racial degeneration, insanity is increasing rapidly, and this, perhaps, is worthy of a moment's consideration. Years ago it was thought to be more of a disgrace to go to an asylum or sanitarium than it is today; the insane are now infinitely more humanely cared for than formerly, and there is less fear among the relatives about having one of their family leave home; therefore, many are now available for statistics which were formerly missed. The horrors that existed in the so-called mad-house are familiar to us all, patients were chained to the walls, they were otherwise abused, and death frequently resulted from accidents, houses of detention were unsanitary, and many died because of such unhygienic conditions.

I think it probable that there is a slight relative increase in the number of insane, and this, in a measure, may be accounted for through our present scientific care of weaklings; thus, children are kept alive today who years ago could not have lived, and consequently we have the survival of the unfit, some of whom are imbecilic, epileptic, degenerate, and insane.

A Comparison of the Onset and Character of the Apoplexy Caused by Cerebral Hemorrhage and by Vascular Occlusion

WILLIAMS B. CADWALADER, M.D.
Instructor in Neurology and Neuropathology at the University of
Pennsylvania, and Clinical Assistant at the Orthopedic Hospital
and Infirmary for Nervous Diseases
PHILADELPHIA

A COMPARISON OF THE ONSET AND CHARACTER OF THE APOPLEXY CAUSED BY CEREBRAL HEMORRHAGE AND BY VASCULAR OCCLUSION*

WILLIAMS B. CADWALADER, M.D.
Instructor in Neurology and Neuropathology at the University of Pennsylvania, and Clinical Assistant at the Orthopaedic Hospital and Infirmary for Nervous Diseases
PHILADELPHIA

INTRODUCTION

The differentiation during life between spontaneous intracerebral hemorrhage and cerebral softening consequent on vascular occlusion is notoriously uncertain. Nevertheless, it is generally admitted, whether correctly or incorrectly, that the mode of onset and character of the apoplectic attack may be sufficient to determine the nature of the underlying lesion.

Thrombosis, like hemorrhage, is dependent on arteriosclerosis or syphilitic arteritis, though some cases of vascular occlusion can be ascribed to certain changes in the blood, as in pneumonia and typhoid fever. Without trauma healthy blood-vessels never rupture. This may account for the rarity of cerebral apoplexy in the lower animals. Dr. Herbert Fox, director of the pathologic laboratory at the Philadelphia Zoological Gardens, has informed me that he has never observed arteriosclerosis of the brain or its results—softening or hemorrhage—in a series of more than twenty-three hundred post-mortem examinations of a large variety of mammals, birds and reptiles.

Disturbance of consciousness is the most characteristic feature of apoplexy. Gowers[1] considered a patient to be unconscious or to have lost consciousness when there was no longer spontaneous evidence of

---

*** From the Laboratory of Neuropathology at the University of Pennsylvania, Department of Medicine.**

[1] Gowers: Text-Book, Diseases of the Nervous System, 1907, ii, 99.

mental action and none could be elicited by sensory stimulation. The exact mechanism concerned in producing this condition is not clearly understood; it is reasonably certain, however, that the preservation of consciousness depends on the proper regulation of intracranial pressure with an adequate supply of blood to the cerebral cortex.

It is believed that the intracerebral pressure may vary slightly under normal conditions, but according to Geigel[2] it must at all times equal the difference between the arterial pressure and the vascular tension, when the arterial pressure remains the same. An increase of vascular tension leads to diminution, and a cessation of vascular tension to increase of intracerebral tension. It is not the quantity of blood constantly present in the cranial cavity, but the quantity propelled in a given unit of time through the vessels of the brain, which determines the nutrition and proper function of the living cerebral substance. When intracerebral pressure increases, the veins and capillaries having very thin walls are necessarily compressed, thus a deficiency in the circulation of the brain is produced. Hence, even a decrease of arterial tension while the arterial pressure remains the same represents a causative factor in the production of the attack.

Anything which brings about a disturbance between the normal relationship of intracerebral pressure, arterial pressure and vascular tension, may cause an apoplectic attack; moreover, it is probable that the character of the attack can be directly influenced not only by the degree of disturbance, but also by the rapidity with which the normal neutralization of these forces is disarranged. Obviously, both hemorrhage and thrombosis can produce such disharmony in relationship and regulation of the forces concerned, although perhaps not in exactly the same manner; still the end-results may be similar.

Thomas,[3] in his article on apoplexy in Osler's "System" quotes von Monakow, who gives the following analysis of the various effects of hemorrhage into the brain: (1) the primary local destruction of the nervous tissue; (2) the edema in its neighborhood; (3) the decrease in the amount of blood in the vessels in the neighborhood of the clot; (4) the accompanying circulatory changes in the cortex; (5) the transmission of the mechanical shock caused by the sudden escape of blood into the brain in all directions throughout the intracranial cavity (this shock besides its physical action, is a physiologic stimulus to various parts of the brain; and especially, von Monakow thinks, it may stimulate the vasomotor centers and cause a reflex contraction of the cortical vessels); (6) increase in intracranial pressure; (7) interference with the circulation of the cerebrospinal fluid. Disturbance of consciousness, he believes, depends on a sudden cerebral anemia, and that this is brought about by some or all of the various factors enumerated above, but that probably the mechanical stimulation of the vasomotor centers is the most important.

---

[2] Geigel: Text-Book, Modern Clinical Medicine, Diseases of the Nervous System, 1908, p. 259.

[3] Thomas: Osler's Modern Medicine, vii, p. 382.

All explanations of the real mechanical changes which do take place within the brain at the time of the initial onset of an apoplectic attack must be somewhat imperfect; still a proper theoretical conception of the various principles concerned is of great assistance in understanding the character of the clinical manifestations of the different conditions under discussion.

In order to ascertain to what extent differences in the character of an attack can be relied on for diagnostic purposes, seventy-two cases of cerebral hemorrhage and fifty of softening have been studied. In each case a post-mortem examination of the brain was made. In addition, the histories of more than three hundred non-fatal cases of hemiplegia were examined, in which an acute vascular lesion had undoubtedly been the initial source of paralysis, but only one hundred of these were found satisfactory for analysis. On account of the fact that so many patients suffering with apoplexy are not observed during the beginning of the attack, many being picked up on the street and brought to the hospital in an unconscious state, the difficulty of obtaining an accurate history is very great; therefore, a large number of cases had to be discarded, and only thirty-eight cases of hemorrhage and twenty-four of softening were made use of, for the accuracy of the statements obtained seemed to be beyond all possible doubt.

In order to facilitate comparisons, cases of hemorrhage and softening have been grouped according to the rapidity with which consciousness was lost.

GROUP A, IN WHICH CONSCIOUSNESS WAS LOST SUDDENLY

This group comprises twenty-four cases of hemorrhage and eleven of softening, each of which began with sudden profound loss of consciousness. These patients were variously occupied pursuing their daily vocations, and were instantaneously struck down totally oblivious of their surroundings. Premonitory symptoms were not recorded. Vomiting occurred early in two cases of hemorrhage, but was absent in each of eleven cases of softening. Violent general convulsions were observed in three cases of hemorrhage and in three of softening. In two of the former cases blood had filled both lateral ventricles, and the convulsions were not only severe, but rapidly repeated until death occurred. In one case of hemorrhage, severe but infrequent convulsions were noted, and the lateral ventricles were not filled with blood; in seven cases the lateral ventricles were filled with blood, yet convulsions did not occur. In two of the cases of softening in which convulsions took place, the basal ganglia alone were affected; but in another case of softening the lesion was confined to the cortex, yet the patient had violent bilateral general convulsions. In every case of this group, whether with hemorrhage or vascular occlusion, the loss of consciousness was equally profound, and except for momentary remissions it was not regained. Hemiplegia was invariably present. In twenty-three of the twenty-five cases the hemorrhage was situated in the region of the basal ganglia and internal capsule, and in nine the lateral ventricles were filled with blood. Only one hemorrhage was small, measuring 5 by 2.5 cm. This was situated directly under the cortex of the right temporal lobe. Each one of the softenings belonging to this group were found in the region of the basal ganglia and internal capsule. Nine were extensive, while only two were small, measuring from 3 to 4 cm. in diameter. In each case thrombosis has been the cause.

The duration of life varied considerably. Only four patients with hemorrhage lived less than one day, one died in six hours, and one survived twenty-one days; but in this latter case the hemorrhage was small and close to the temporal cortex of the right side and death may have been hastened by erysipelas. Of the patients with softening, one died after twelve hours and one lived sixteen days, the average being four days. These results seem to be in accordance with Spiller's[4] observations.

GROUP B, IN WHICH CONSCIOUSNESS WAS NOT LOST SUDDENLY

The second group includes those cases in which the initial disturbance of consciousness was rather mild and then gradually increased till in about ten or fifteen minutes or longer the stage of coma was reached. Partial recovery frequently occurred for short periods of time. There were eleven hemorrhages and only three softenings with this type of onset. Premonitions were recorded in each case of the softenings as well as in five of the eleven hemorrhages. Vomiting occurred only once and then with hemorrhage. General convulsions did not occur in any case, whether of hemorrhage or softening, but in one case of hemorrhage convulsive movements of the upper extremities preceded impairment of consciousness by a few minutes. In every case hemiplegia was present. Twice hemorrhage originated from a cortical vessel, and a large mass of blood was found covering much of the cerebral cortex. As in the preceding group, hemorrhage occurred in the region of the internal capsule and basal ganglia nine times, but the hemorrhages differed from those of the former group in that they were smaller and more localized and blood had not filled the lateral ventricles. Only two were really small, however, one measuring 2 by 2 cm. situated within the lenticular nucleus and implicating about 1 cm. of the internal capsule, and one 3.5 by 3.5 cm., within the frontal lobe near the cortex.

Of the softenings, three had been very gradual in onset. Hemiplegia and aphasia were established slowly, requiring some days previous to the impairment of consciousness. In these cases the left sylvian artery has been occluded and a large part of the cerebral cortex on the left side was destroyed, including the motor convolution, temporal convolution and parietal lobe. There were three cases of hemorrhage in which motor weakness preceded disturbances of consciousness, but only by a few minutes and not by hours or days, as in some softenings.

---

[4] Spiller: Univ. of Penn. Med. Bull., October, 1906.

The average duration of life was six days in this group of hemorrhages; one patient lived eleven days and one died in three days. Unlike those of the previous group, none died during the first twenty-four hours. Each of the three patients in whom extensive softening had occurred lived for five days after consciousness was lost, but motor weakness had preceded the attack by some time, exactly how long could not be determined, still at least four days in one instance. In four cases of softening consciousness had not been disturbed in the least; but each of these had been caused by an embolus of the middle cerebral artery during an acute disease; a large part of the central portion of one hemisphere was destroyed, including all the basal ganglia and part of the temporal lobes. Two patients lived more than a month, and death was probably caused by cardiac failure and not by the cerebral lesion. In one instance of hemorrhage confined to the pons, consciousness was not impaired until the fifth day after paralysis had occurred, and even then it was not lost until a few hours before death.

HEMIPLEGIA

For the purpose of comparison one hundred cases of hemiplegia have been studied. All of these patients were seen at the Orthopaedic Hospital and Infirmary for Nervous Diseases, and were believed to have had an acute vascular lesion of the brain, that is, thrombosis, embolism or hemorrhage. The period of time which had elapsed after the original apoplectic attack and time of the physical examination varied considerably; the average, however, was five or six months.

Thirty-two of these patients complained of various degrees of ill health preceding the date of the onset of apoplexy; headache was described in nine cases, and syphilis was evident in thirteen. The attack began during the course of an acute illness seven times. In only three cases were there more or less definite sensations of numbness and tingling with some pain and discomfort spoken of as occurring on the side of the body which shortly became paralyzed.

In fifty-two cases consciousness was said not to have been disturbed even in the slightest degree, but these patients did complain of various sensations, such as headache, vertigo, weakness, nausea, faintness, etc., which are too indefinite to be classified, but which might be collectively considered as the "apoplectic equivalent." Hemiplegia developed during sleep in twelve cases.

Consciousness was lost suddenly before any signs of paralysis were noticed in seventeen cases, but in five cases paralytic signs developed first and were then followed by a sudden loss of consciousness. Slow progressively increasing loss of consciousness was rare, occurring only three times, twice preceding any sign of weakness, while in the one case definite impairment of function was the first indication of disease.

Convulsive movements preceded paralysis by some hours in two cases, but general convulsions were not recorded. Aphasia was recorded in nine cases.

The character of the paralysis in every case was the ordinary type of hemiplegia or hemiparesis, with increased tendon reflexes. Sensation was impaired in eight cases, and the face entirely escaped four times.

Many writers have formed the habit of describing premonitory symptoms, which are of value in differentiating hemorrhage, thrombosis and embolism. On purely theoretical grounds it is almost inconceivable that a miliary aneurysm or an arteriosclerotic vessel within the brain in the act of bursting can give rise to symptoms which could be separated from and not confused with the various phenomena of the early stage of the rapidly oncoming apoplectic state. On the other hand, there undoubtedly exist many cases of vascular occlusion in which the lumen of the vessel is cut off gradually, causing thereby gradual impairment of function of that part of the brain nourished by the vessel affected. Chiefly on this ground the statement is often made that premonitory symptoms are absent in cases of hemorrhage and frequently present in cases of thrombotic softening.

Arteriosclerosis or syphilitic arteritis is the common forerunner of most cases of apoplexy. Patients so affected frequently complain of various sensations referable to cerebral disease—headache, vertigo, numbness and tingling of one or more extremities, and a variety of subjective disturbances. These are the symptoms so frequently referred to as premonitions immediately preceding an attack. It must be remembered that when an apoplectic attack begins, the loss of consciousness may develop gradually or suddenly. In the latter case, the starting-point of the attack is unmistakable; but, whenever consciousness is lost very gradually, many symptoms may develop which are in reality caused by the acute vascular lesion, and are indeed actually nothing more than the first and earliest manifestations of a gradual transition from the conscious to the unconscious state. Many apoplexies begin with vertigo, headache, confusion or disorientation, which phenomena may more or less rapidly progress till the grade of profound coma is reached. Taking this point of view, it is evident that symptoms of chronic vascular disease of the brain or arteriosclerosis cannot be distinguished clinically from the earliest symptoms of some cases of apoplexy regardless of the nature of the lesion causing it. This fact is only too well illustrated when it is remembered that some arteriosclerotic patients suffer from apoplectiform attacks which are not associated with cerebral softening or hemorrhages, and which differ only in degree and not in the essential characteristics of the attack itself from true apoplexy.

From what has been said, the so-called premonitions should be more frequently observed whenever consciousness is disturbed at first slowly and therefore incompletely, and less frequently when it is disturbed suddenly and severely.

Many years ago it was said by Trousseau (quoted by Thomas,[3]) that sudden loss of consciousness is rarely if ever the initial symptom of apoplexy. But physicians seldom have the opportunity of observing the beginning of an attack, so that much judgment is required in interpreting the descriptions given by the patient or friends. I was once present where an elderly lady was quietly sitting at the dinner table carrying on an animated conversation—curiously enough, with two physicians—when suddenly she dropped knife and fork from her grasp and fell backward in her chair totally unconscious; some hours later she was dead. If premonitions had been present, they would surely have been noticed.

Of the thirty-eight cases of hemorrhage which have been studied here, premonitory symptoms of short duration were recorded five times. In one there had been a feeling of excitement and apprehension lasting fifteen minutes, accompanied by convulsive movements of both arms. Numbness and tingling of all four extremities and headache occurred once; general restlessness and excitement were present once; while in two instances there was a feeling of intense general weakness. In each case these symptoms were the first symptoms of cerebral hemorrhage, and were recognized only because consciousness was lost gradually. In one case of softening the patient complained of numbness of one side, and three developed motor weakness before the attack; in each consciousness was gradually lost.

RELATION OF THE SIZE OF THE LESION TO SEVERITY OF ATTACK

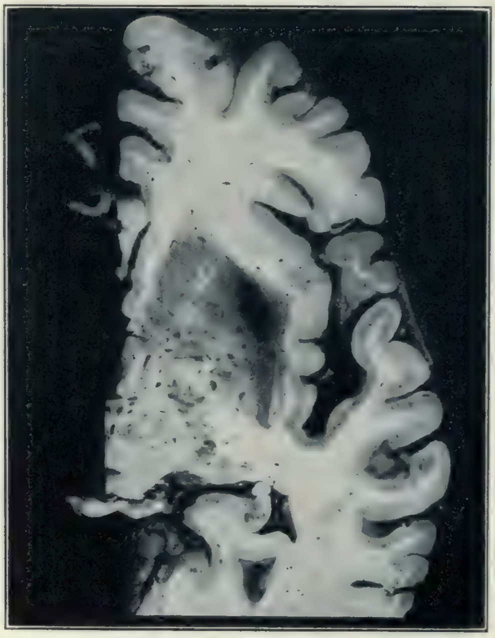
As already mentioned, the hemorrhages of the first group, with only one exception, were large, and in all the initial apoplexy was sudden and severe. Those of the second group were smaller, though only two were really small; the smallest of all measured 2 by 2 cm. In these apoplexy was less abrupt than in the former group. Naturally hemorrhages may vary greatly in size. Cases have been observed in which nearly all of one hemisphere and part of the other have been destroyed, and many are seen as small as an English walnut. Small hemorrhages, however, are exceedingly uncommon—that is to say, those as small as a pea. Attention has been called to this fact by von Monakow.[5] The cases studied here would indicate that large hemorrhages are most common and also that the sudden severe types of apoplexy usually result. I know of no instance in which a large spontaneous hemorrhage within the brain did not produce loss of consciousness, though the initial onset may not have been sudden. Even a moderately small hemorrhage may cause a severe and sudden attack, as in one case recorded in the first group. It seems correct to conclude that the size and rapidity of growth of the hemorrhage may have a very decided influence on the severity of the initial apoplexy. No doubt Gowers'[6] statement that "the intensity of apoplexy is a graver indication than its duration" can be explained on those grounds, for the duration of life is apt to be longer when hemorrhage is small. The cases of my first group seem to corroborate this statement. Of softening from thrombosis or embolism, in a measure the same statements are true, but not so regularly.

---

[5] Von Monakow: Gehirnpathologie, 1897, p. 97.

[6] Gowers: Text-Book of Nervous Diseases, 1907, ii, 443.

Fairly large softenings are occasionally found at necropsy which had not caused very distinct apoplexy during life. Indeed, there are four cases already referred to in which the middle cerebral artery had been occluded, two by embolism, during the course of an acute endocarditis, and twice by thrombosis during pneumonia. In each a large portion of the basal ganglia and adjoining structures were destroyed, yet a very indistinct apoplectic attack was observed during life. The smallest hemorrhage of which I have knowledge is shown in the photograph given here. It occurred in a patient in the service of Dr. Charles K. Mills at the Philadelphia Hospital, and the specimen is in Dr. Spiller's laboratory. Unfortunately the history of


Very small intracerebral hemorrhage which caused death.

this case is incomplete, but it is probable that there had not been a very distinct apoplectic attack, consciousness was not disturbed early. On the other hand, there were two softenings recorded in the first group measuring only from 3 to 4 cm. in diameter, with distinct apoplexy. Again Gowers[7] has said, "of lesions of equal size the more sudden the obstruction the more pronounced the attack." I would accept the statement as correct in the majority of instances, and would further enlarge on it by adding that the more gradually vascular occlusion is produced the less pronounced the attack or the more gradual the onset. This is well illustrated by the cases mentioned in the second group, in which the sylvian artery had become thrombosed; in each of these cases there had been a very gradual and progressive development of paralytic symptoms with little disturbance of consciousness. Spiller[8] has called attention to this type of case in distinction to the gradually increasing hemiplegia of cerebral tumor. In this connection it should be remembered that when a vessel is suddenly occluded, apoplexy may immediately result, together with paralysis, but actual softening of the brain requires at least a day to develop after occlusion takes place. I have been able to prove this to my own satisfaction by experiments on dogs.

It is difficult to explain the very sudden loss of consciousness that occurs in some cases of cerebral softening; a rapid onset, however, suggests that the affected vessel is occluded suddenly. Cases of so-called angiosclerosis which have been carefully studied with the ophthalmoscope seem to furnish considerable evidence to strengthen the belief that cerebral vessels may be affected like the arteries of the retinae, which become instantaneously and permanently occluded. In some instances the retinal vessels have been watched while being occluded.

This subject has been recently discussed by Taylor, Werner and Coates.[9]

Assuming that coma when caused by hemorrhage was due entirely to increased intracranial pressure, Cushing[3] (quoted by Thomas) proposed opening the cranium and evacuating the clot. There can be no doubt, that when hemorrhage is fairly large, the intracerebral pressure is greatly increased and that this is one of the most important factors producing coma and death, for it is not uncommon to find at necropsy that the hemisphere in which hemorrhage has occurred is distended and larger than the opposite or sound side. In a recent communication Marie[10] stated that coma is due much less to the sudden eruption of a large mass of blood into the affected hemisphere than to the compression of the brain caused by the presence of this quantity of blood. The sudden eruption of the blood produces ictus, which is a transitory phenomenon; the compression of the brain produces coma, which is a persistent phenomenon; but a deep and lasting coma is not produced unless the opposite hemisphere is compressed also. He was able to demonstrate that hemorrhages measuring as much as from 5 or 6 cm. by 3 or 4 cm. did not produce coma unless there was compression of the opposite side. When the unaffected hemisphere was compressed, the coma was profound and absolute. As a result of these observations, Marie advocates trephining on the sound side in order to relieve pressure. In four instances his results were good, but he does not give histories of his cases.

---

[7] 7. Gowers: Text-Book of Nervous Diseases, 197, ii, 432.

[8] 8. Spiller, William G.: Brain Tumor, The Journal A. M. A., Dec. 18, 1909, p. 2078.

[9] 9. Taylor, Werner and Coates: Tr. Ophth. Soc. U. Kingdom, 1913, xxxiii, pp. 1, 9, 30.

Marie's views would seem to be logical. In certain cases operation might be beneficial, for without operation death cannot be prevented.

CONCLUSIONS

It is thoroughly appreciated that "generalizations may be dangerous," for exceptions always occur. Nevertheless, the following conclusions would seem to be warranted:

Spontaneous intracerebral hemorrhages are apt to be large; very small hemorrhages are rare. Of seventy-two specimens examined, only four measured less than 4 cm. in their broadest diameter.

It is certain that large hemorrhages are always fatal, and it is also certain that small hemorrhages may be fatal, and it even seems probable that hemorrhages are always fatal, no matter whether small or large.

---

[10] Marie: Bull. d. l'Acad. de méd., Paris, Nov. 18, 1913, p. 405.

When repeated attacks of apoplexy with hemiplegia occur in the same patient at different times, the final or fatal attack may be due either to softening or to hemorrhage, but the former non-fatal attack is invariably caused by vascular obstruction and softening and not by hemorrhage. Repeated attacks of intracerebral hemorrhage are not compatible with life.

Small and moderate-sized lesions within the brain, generally described as cysts, are apt to be considered the result of vascular occlusion; but in some instances such lesions may be produced by hemorrhage which has become healed. Their true origin in some cases seems uncertain, but they have been classified here as softenings.

The duration of life is generally longer with small hemorrhages than with large ones. Sudden death within a few minutes after the onset of apoplexy does not occur even though the lesion is a large one. It is remarkable that fairly large hemorrhages may not in all instances cause rapid death. Spiller[6] has recorded a case in which a clot was found partly encapsulated and measured 7 by 2.5 cm., yet the patient lived almost two months.

The type of apoplexy produced by hemorrhage and by vascular obstruction is not of a distinctive kind. The onset and character of the apoplexy may be exactly alike, though the lesion is entirely different. But a sudden onset with rapidly developing and persistent coma usually indicates hemorrhage. A slow onset with premonitory symptoms without profound coma may be due to hemorrhage or to softening, but the less severe the disturbance of consciousness the more likely that it is caused by softening and not by hemorrhage.

Premonitory symptoms are not characteristic of the lesion; as a general rule they are recorded in the milder types of apoplexy in which the onset is not abrupt.

Slowly increasing loss of consciousness ending in profound coma, known as ingravescent apoplexy, is generally due to hemorrhage.

It is doubtful if hemorrhage ever occurs without causing very distinct disturbances of consciousness, but it is certain that many softenings do occur without producing distinct apoplectic attacks. Most non-fatal cases of hemiplegia are caused by vascular occlusion and subsequent softening. The mere fact that life is preserved is in itself indicative of the absence of hemorrhage.

The type of apoplexy probably depends more on the size of the hemorrhage than its situation, but with softening the rapidity with which the vessel is occluded may influence the rapidity of onset of the attack as well as the extent of the lesion.

I am indebted to Dr. Allen Smith for the photograph and I also wish to express my thanks to Dr. Spiller, who very kindly allowed me to use the specimens in his laboratory, and to Dr. Warfield T. Longcope for the material obtained in the Ayer Clinical Laboratory of the Pennsylvania Hospital, and also to the Medical Staff of the Orthopaedic Hospital and Infirmary for Nervous Diseases for the use of the records of cases examined in their service.

1710 Locust Street.

Reprinted from The Journal of the American Medical Association
May 2, 1914, Vol. LXII, pp. 1385-1389

COPYRIGHT, 1914
American Medical Association, 535 N. Dearborn St., Chicago

Reprinted from the New York Medical Journal for June 14, 1913.

HALLUCINATIONS OF HEARING AND DISEASES OF THE EAR.*

By John H. W. Rhein, M. D.,
Philadelphia,

Professor of Diseases of the Mind and Nervous System at the Philadelphia Polyclinic and College for Graduates in Medicine; Neurologist to the Howard Hospital; Physician to the Philadelphia Home for Incurables, etc.

Recently there have come under my observation three cases of hallucinations of hearing associated with disturbances in the peripheral auditory apparatus, in which the hallucinations were the only manifestation of mental disease. In referring to the literature I find that this condition, while well recognized by some writers, Kraepelin (1), Wernicke (2), White (3), and others, has been infrequently described. Several phases of the subject are of interest, namely, the peripheral origin of hallucinations, the bearing that disease of the bloodvessels of the brain has upon the development of this symptom, and the influence of heredity. In the first place, recognizing that three theories exist to explain the occurrence of hallucinations, namely: 1. The theory of the cortical origin; 2, the theory of the subcortical origin; and, 3, the theory of the peripheral origin, the association of hallucinations of hearing with aural disease in individuals otherwise psychically healthy, has a bearing on the study of hallucinations as relating to the theory of their peripheral origin. It may be stated in passing that the first two are acknowledged to be the most plausible of these theories.

While in this paper the object is not to discuss at length the various theories advanced to explain the physiological pathology of hallucinations, I

---

*Read before the Philadelphia Psychiatric Society, March 14, 1913.

Copyright, 1913, by A. R. Elliott Publishing Company.

cannot escape a reference to some features bearing upon their peripheral origin. While some authors deny that hallucinations are ever of peripheral origin, Tanzi (4) and Tamburini (5), there can be no doubt that irritation of the peripheral sensory organ plays some part in the causation of some hallucinations. Thus, there are numerous examples on record of hallucinations of sight related to diseases of the optic nerve, Briand (6), to injury of the cornea, Naegeli (7), and to chorioiditis with central scotoma, Uhthoff (8). Furthermore, hallucinations of sight have resulted from pressure on the eyeball in alcoholics, a phenomena described by Liepmann (9). Again, electrical stimulation of the acoustic nerve has caused hallucinations of hearing, Jolly (10) and Moravcsik (11). In addition, there are on record a few cases which seem to demonstrate more than an accidental relation between irritation in the sphere of the peripheral apparatus of hearing and hallucinations of hearing. Stein (12) described the case of a man of seventy-eight years, who heard words in association with melodies occurring in the right ear, which disappeared when a plug of cerum was removed. Further examination showed that the psyche of the patient was changed and that he showed symptoms in the nature of a slight *Beziehungswahn*. Stein believed there was a sclerotic senile change in the gray substance of the brain in the region of the word centre.

Klieneberger (13) reported three cases which are sufficiently interesting to cite in abstract. The first was that of a woman of fifty years who suffered with middle ear disease. The right ear drum was retracted and was the seat of a scar. She heard voices of a threatening character. The voices located themselves first in the room and later in the ear. These hallucinations were preceded by various sounds which were in the nature of hissing and the like. There were no other symptoms of mental disorder, and the voices, though at first influencing her conduct, later were disregarded.

There was a history of nervous disease in the ancestry. The second case was that of a man of fifty-three, without history of hereditary taint, who, following an accident, suffered from neurasthenic manifestations and later heard voices calling him by name and insulting him. The voices disturbed him, causing depression and disquietude. In one ear the voice was that of a man, in the other of a woman. There were no delusions. There was a catarrh of the Eustachian tubes, and the ear drums were retracted. Under treatment the voices disappeared, though his power of comprehension suffered. The third case was that of a woman of fifty-two with a negative family history who had sixteen years previously suffered from an attack of hypochondriasis from which she recovered entirely. She heard voices and whistling in both ears, later singing in the left ear, then the right, and finally sounds like a harmonica, a trombone, and a phonograph. Melodies and songs of her childhood days which she had forgotten were heard. These were more noticeable when not working and alone. Her actions were uninfluenced by them. She was otherwise, psychically as well as physically, intact, except for a chronic change in the ear drums and catarrh of the Eustachian tubes. All his patients were old people in whom the symptoms indicated a suspicion of disease of the arteries of the brain.

Hudovernig (14) placed on record an interesting case of a lad of eighteen who, besides akoasms, heard unpleasant voices (phenomes) which caused anorexia and insomnia. The patient had a neuropathic history and was himself neuropathic. There was a large amount of wax in the external auditory meatus which impinged upon the ear drum, and after this was removed the tinnitus and hallucinations gradually disappeared. Regis (15) published a report of a case in which the patient, suffering from otitis, first heard simple noises, then hallucinations, and finally delusions of persecution developed. He called attention to the fact that the relation of hallucination and disease of the ear had been already described by Séglas, Bechtereff, and others.

Most of the text books on diseases of the mind and diseases of the ears neglect to mention this subject. However, Kraepelin called attention to the frequency of hallucinations in ear disease, and concluded that there existed some association between the two conditions, and Wernicke stated that in cases of unilateral hallucinations of hearing, which he believed were not rare, there was usually more or less diminution of hearing. He cited a case illustrating this association. Tanzi, who believes that hallucinations of peripheral origin do not occur, looks upon the production of elementary images in consequence of excitation of peripheral nerves such as develops as the result of pressure upon the eyeball, causing luminous objects, or the occurrence of musical sounds in otitis, and hallucinations following galvanism of the acoustic nerve, as perfectly normal occurrences, and outside the domain of psychiatry. There is according to Klieneberger, Forster (16), and Bonhoffer (17), reason to believe that a cerebral arterial sclerosis has an important bearing upon these cases. Apropos of arterial changes in the brain, Ballinger (18) described, as symptoms of arterial sclerosis of the labyrinth, unilateral or bilateral tinnitus, with progressing deafness, impairment of air and bone conduction, dizziness, and, later, hallucinations of hearing. McBride (19) refers to auditory hallucinations, such as hearing melodies and human and animal voices, in cases of ear disease in which insanity could not be suspected, while Knight and Bryant (20), on the contrary, describe this association in the insane.

The question arises whether it is proper to speak of these as hallucinations at all, in the strict interpretation of the term. If an hallucination is a perception without a sensory impression, would it not be proper to look upon such phenomena really as illusions, inasmuch as there is actually a sensory sensation always in these cases? However, whichever position we may take, it does not really detract from the importance or significance of the phenomena as indicative of mental disease.

The following cases illustrate several features of this discussion.

Case I. A woman of fifty with negative family history suffered from fifteen to twenty years with attacks of vertigo and noises in the left ear. About a year before coming under observation at the Philadelphia Polyclinic Hospital in November, 1912, she imagined people were talking about her. She could hear the voices saying, "Slop boy," "Granny," "Grandmother," "Cheese it!" The voices were referred to the school nearby and to the houses of neighbors. She believed that the neighbors incited the boys to annoy her. At first the voices disturbed her, but more recently the voices at times disappeared and ceased to upset her. She talked at times unintelligently to her husband, but otherwise the examination was negative as to delusions, orientation, and other psychic phenomena. She slept well, her appetite was good, and she was able to do her work properly. She had been for some time previous to the onset of the hallucinations deaf in her left ear.

Case II. A Jewish woman of sixty-five applied for treatment at the Philadelphia Polyclinic Hospital. The family and previous histories were negative. For four years she had heard voices expressing themselves in songs. These songs were those of her childhood days. At first there was only one song, but within a few weeks previous to her coming under observation many songs of her childhood days were heard. These songs were located in her ears, that is to say, they did not seem to emanate from without. While the songs disturbed her sleep and were annoying, they did not affect her conduct of life. There were no other mental symptoms that could be elicited. She complained of general tenderness of the subcutaneous fat, more marked in the arms, over the triceps, and also in the fat of the thighs and sides, suggesting adiposis dolorosa. The examination of the nervous system, as well as that of her heart, lungs, and other organs, was entirely negative.

The examination of the ears made by Dr. George W. Coates showed the following: There was some nerve degeneration accounting for the deafness. There was a slight retraction of the ear drums, and some catarrhal condition of the middle ears. The Eustachian tubes were patulous, and the labyrinths reacted readily to the caloric test, showing that they were functionating.

Case III. A woman of sixty-nine, whose father and mother died of apoplexy and two sisters of phthisis. Outside of having had a fibroid tumor, which was removed twenty-five years ago, the patient was apparently well until twelve years ago, when she had an epileptic convulsion. She suffered from a number of these attacks until three years ago; since when she had had no attacks whatever. She had had a progressive catarrhal condition of the middle ear, with retraction of the ear drums for several years. Her condition was briefly as follows: There was a marked anterolateral curvature of the spine due to spondylosis rhizomylique. She also complained of pain in the right knee joint and in both hips. Otherwise the physical and nervous examination was negative. An examination of the ears showed retraction of both drums and marked deafness on both sides. There had been for some years ringing and roaring in both ears which progressively increased. In the last few months she had heard voices in her right ear. These voices said over and over again, "Walk in," "Walk in." The voices also said other things, but these she could not recall at the time of the examination. Her mental condition in all other respects was entirely normal.

In all three cases described the possibility of arterial change in the brain must be recognized and especially in the last two patients, whose ages were, respectively, sixty-five and sixty-nine. It is difficult to avoid the conclusion that the nutrition of the auditory centres has been implicated in these cases and that, therefore, they react abnormally to peripheral irritation. While the occurrence of hallucinations of hearing associated with disturbances of the peripheral auditory apparatus does not necessarily indicate the onset of insanity, there can be little doubt that this phenomenon is an expression of an unstable psychic equilibrium and is therefore actually within the domain of psychiatry. A history of nervous disease in the ancestry is usual, and a study of the cases shows a tendency at some time to exhibit neuropathic manifestations.

REFERENCES:

1. KRAEPELIN: *Psychiatrie*, 1889, p. 28. 2. WERNICKE: *Grundriss der Psychiatrie*, 1906, p. 182. 3. WHITE: *Outlines of Psychiatry*, 1913. 4. TANZI: *Mental Diseases*, 1909. 5. TAMBURINI: Ballet, *Revue neurologique*, 1896, p. 472. 6. BRIAND: *Annales de médecine psychologique*, 1897, v, p. 268. 7. NAEGELI: In Liepmann, *Archiv für Psychiatrie*, 1895, xxvii. 8. UHTHOFF:

Monatsschrift für Phychiatrie und Neurologie, 1899, p. 247. 9. LIEPMANN: *Archiv für Psychiatrie*, 1895, xxvii, p. 172. 10. JOLLY: *Ibidem*, 1873-4, iv, p. 495. 11. MORAVCSIK: In Goldstein, *Ibidem*, 1908, p. 584. 12. STEIN: *Jahresbericht für Neurologie und Psychiatrie*, xi, p. 1118. 13. KLIENEBERGER: *Allgemeine Zeitschrift für Psychiatrie*, 1912, lxvi, p. 285. 14. HUDOVERNIG: *Centralblatt für Nervenheilkunde und Psychiatrie*, 1906, p. 255. 15. REGIS: *Journal de médecine de Bordeaux*, 1904, p. 541. 16. FOERSTER: *Allgemeine Zeitschrift für Psychiatrie*, 1909, lxvi, p. 915. 17. BONHOFFER: *Ibidem*, lxvi, p. 915. 18. BALLINGER: *Diseases of the Throat and Ear*, 1911, p. 207. 19. McBRIDE: *Diseases of the Throat, Nose, and Ear*, .1894, p. 422. 20. KNIGHT and BRYANT: *Diseases of the Nose, Throat, and Ear*, p. 429.

1732 PINE STREET.

[Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 41,
No. 6, June, 1914.]

EXTENSIVE BILATERAL SUBDURAL HEMORRHAGE OVER BOTH CEREBRAL HEMISPHERES WITHOUT MOTOR PARALYSIS OR IRRITATION

By William G. Spiller, M.D.

The patient was a man aged 52 years, who came into the service of Dr. Spiller at the Philadelphia General Hospital January 13, 1913. He stated that on December 13, 1912, he, with another man, was pitching bales of paper onto a wagon; he had pitched one bale to a height of about fourteen feet and had turned his back when the bale fell and struck him on the top of the head. He estimated the weight of the bale as about one hundred and twenty pounds. He was not knocked down but he thought his neck was broken, and he was dizzy. He did not lose consciousness but was obliged to sit down because of dizziness. His neck felt stiff and he was unable to continue his work. At the time of admission, one month after the accident, he could use his upper and lower limbs in a normal manner and had no disturbance of bladder or bowels. He had been continually drowsy since the accident and had dreams at times of falling. The appetite was poor but the bowels were regular. He had vomited almost daily since the accident and had dyspnea, but had not had convulsions and had no tremor. He had used much alcohol. There was no paralysis of any part of the body, the ocular movements and the iridic reflexes were normal. The pulse was slow and full. The tendon reflexes of the upper and lower limbs could not be obtained, and there was no upward movement of the big toes in testing for the Babinski reflex. No ataxia could be detected. He complained much of headache, especially when the head was held backward to test the iridic reflexes, and he had some photophobia.

The important features of this case were: history of a severe blow upon the head, followed by continued drowsiness, vomiting, headache and loss of tendon reflexes in an alcoholic man. There was no indication of paralysis or irritation of either motor area of the brain, and at the time the man came under Dr. Spiller's care this condition had existed one month.

An X-ray photograph of the head showed no fracture. An ophthalmoscopic examination could not be obtained immediately, but when it was obtained the report was bilateral low grade optic neuritis, more distinct on the left side. The irides reacted to light and in convergence. The man's condition when this report was received was too severe for operation. He had been unconscious for three days but moved both upper limbs voluntarily. The head could be moved passively from side to side without rigidity of the neck, but this movement caused an expression of discomfort. The pulse, temperature and respiration remained normal until two days before death.

At the necropsy the skull showed no fracture. An extensive hemorrhage covered the frontal, motor and parieto-occipital regions on each side.

Operation had been considered but the long duration of symptoms of a mild type before the patient entered the hospital, the knowledge that the man had used alcohol freely, the absence of fracture of the skull made hesitation desirable, and when the ocular report was received the stupor was so great that operation probably would have been useless.

It is hard to believe that this great subdural hemorrhage had existed four to six weeks without causing more symptoms than were present in this case; it is equally hard to believe that a moderate hemorrhage at the time of the accident was followed by a severe hemorrhage six weeks later. The absence of paralysis or convulsions is remarkable. The drowsiness, vomiting, headache and loss of tendon reflexes persisting from the time of the accident until death make the existence of hemorrhage dating from the accident probable. It is possible that some weakness of the limbs occurred just before death during the stupor.

The question of subdural hemorrhage without fracture of the skull is an extremely important one and was discussed by the writer in the International Clinics, Vol. IV, 1903. It has been discussed recently in an exceedingly interesting paper by Bychowski, *Zeitschrift für die gesamte Neurologie und Psychiatrie*, Vol. XIV, No. 3, 1913.

[Reprinted from The Journal of Nervous and Mental Disease, Vol. 41, No. 2, February, 1914.]

SILAS WEIR MITCHELL, M.D., LL.D.

His Place in Neurology

By Charles K. Mills, M.D.

The death of Silas Weir Mitchell on January 4, 1914, shortly before he had completed his eighty-fourth year, marked the passing of one of the most striking figures in American medicine. The name of Rush in our nation's early history and that of Mitchell in the chronicles of the last half century stand side by side in estimating medical achievement and position in this country. Born a little more than a half century after the birth of the nation and beginning his professional career a decade before the opening of the civil war, he is conspicuous in the most eventful and progressive period of our national history.

To others must be left the recital of the inspiring story of Mitchell's place in literature, science and civic affairs. In the brief time at my disposal a hastily prepared summary and appreciation of his work in neurology is all that I can hope to present. A more thorough and elaborate study of his neurological contributions is due to his memory and will no doubt be forthcoming in some volume dealing fully with his life work. The numerous honorary degrees bestowed on him by the great universities of his own and foreign countries and his admission to membership in those scientific societies both of America and Europe universally recognized as the bodies most capable of estimating intellectual worth and achievement, were due in no inconsiderable part to his brilliant work in expanding and advancing the data and principles of neuro-physiology and neurology.

It will be impossible in a few pages to even outline and imperfectly estimate the contributions of Mitchell to neurology. These were chiefly along the lines of (1) clinical and pathological observations on injuries and diseases of the peripheral nerves; (2) physiological investigations of the cerebellum; (3) the therapeutics of the nervous system, especially as illustrated by his organization of the rest treatment and by his numerous additions to our knowledge of the use of drugs and of special methods of local treatment; (4) the establishment of new clinical types and the more thorough study of symptoms and clinical combinations recognized but not fully appreciated and expounded by others.

Mitchell received his degree at the Jefferson Medical College in 1850 when in his twenty-first year and soon began his well-known researches in physiology, comparative anatomy and toxicology, carrying these on in such time as he could secure from his work as a practising physician. As is well known, his earliest ambition was to make for himself a place as a physiologist. The study of the venom of serpents, of the action of vegetable poisons and of special points in the physiology of some of the lower animals, claimed much of his time and attention during the first ten years after graduation. While these investigations were not solely, nor chiefly, neurological, they contributed facts of value to the anatomy, physiology and toxicology of the nervous system. An interesting discovery which paved the way for others in the same direction was that of a laryngeal chiasm in chelonia similar to the optic decussation.

Throughout a large part of his long scientific and professional career Mitchell's mind continued to be fascinated by the question of the action of venoms. He recurred at intervals, alone or with others, to new studies in this field of research, his mind grasping the fact that in such investigations would be found data that would help to clarify some of the most obscure problems in physiology, sero-pathology and therapeutics. His latest work in this connection was done with Reichert in the Physiological Laboratory of the University of Pennsylvania, more than a quarter of a century after his first researches.

The events and opportunities of the civil war gave the first strong stimulus to Mitchell's purely neurological investigations and contributions. Early in the war he was appointed an acting assistant surgeon and began his duties in one of the large military hospitals in Philadelphia. Having especial interest in everything which related to the nervous system, and his fellow surgeons in the main inclining to the neglect of neural maladies, Mitchell soon had large opportunities thrown in his way. Wards for the study and treatment of wounds and diseases of the nerves and brain and cord were set apart for him in two large hospitals and in time a suburban hospital known in local and medical history as the Turner's Lane Hospital of Philadelphia, with accommodations for several hundred cases, was almost entirely given over to the study of injuries and diseases of the nervous system, the work along this line being left almost exclusively to Drs. Mitchell, Morehouse and Keen. The special contributions and the monographs which grew out of the work at this hospital are now well known and need not here be recited in detail.

It may add interest to this sketch to give a brief citation from Mitchell's own reminiscences of this early but invaluable experience: "The opportunity was indeed unique and we knew it. . . . Thousands of pages of notes were taken. There were many operations and frequent consultations. About midway we planned the ultimate essays which were to record our work. There was a small book on Neural Injuries full of novelty, a short essay on Reflex Palsies, etc. One of the most notable was Keen's essay on Malingering. Others on Epilepsy, muscular disorders and on acute exhaustion were never written because of accidental destruction of notes by fire."

Out of this work during the time of war proceeded not only the original monograph and separate essays of Mitchell, Morehouse and Keen, but the later expanded and thoroughly systematized volume of Mitchell in 1872 on "Injuries to Nerves and their Consequences" and Dr. John K. Mitchell's book reviewing the remote consequences of these nerve injuries from a study of the condition of some forty of the survivors. Forms of neuritis, trophic disorders and symptoms like causalgia, hitherto undescribed, the physical and psychic phenomena presented by those who had lost limbs through amputation, and various methods of treatment particularly adapted to nerve injury were here, with a wealth of other detail and suggestion, first brought to the attention of the profession. Mitchell's keen interest in the study of peripheral nerve phenomena remained with him throughout his career and led to some of his most important contributions. He was a pioneer in the minute study of cutaneous nerve supply, in this respect in not a few particulars forerunning the work of Head and his collaborators and contemporary investigators. In one of his papers published as early as 1873, he showed that our views as to cutaneous sensory supply, founded largely upon anatomical observations, needed to be thoroughly revised. He found, to use his own words, that surface anatomy was a fiction. Mitchell showed by his own observations, citing also those of Letievant and others, that section of the median or other nerve trunk did not completely annihilate sensibility in distribution of the nerve according to the usually accepted anatomy. He recognized clearly the fact of overlapping nerve areas and the main distinctions between what have since been called protopathic and epicritic sensibility, also various erroneous ideas as to regeneration of nerves and much else that has since been seen and added to by others. His first work in this direction done as early as 1873 was followed by other papers during ten or more years.

The researches of Mitchell on the physiology of the cerebellum placed him in line as an experimentalist with distinguished contemporaries and predecessors in this field. These investigations, with such time as he could command from his duties as a medical practitioner, were continued over a period of six years, from 1863 to 1869. In the résumé of his work and results in the *American Journal of the Medical Sciences* for April, 1869, he shows in the first place that he was thoroughly familiar with all that had been done from the time of Rolando to the publication of his article,—with the work of Flourens, Lussana, Bouillaud, Serres, Wagner, Magendie, Brown-Séquard, Vulpian, Luys, Richardson, Longet, Dalton, and others.

His experiments, which were performed upon pigeons and other birds, and upon rabbits and guinea pigs, were mainly of three sorts, namely, ablation, partial or nearly complete, freezing with rhigoline spray, and injections of globules of mercury into selected portions of the cerebellum. He also produced irritations by directly applying cantharides to exposed parts, or by penetrating the organ with an awl-shaped instrument. He ablated the cerebellum eighty-seven times and performed two hundred and sixty experiments on the influence of irritants. The results of these investigations are of course well known. They were in some measure confirmatory and in some antagonistic to those obtained by others. It was a matter of surprise to me that in the symposium on cerebellar symptoms and localization at the recent London congress, little was said with regard to Mitchell's work. It is true that the view at which he arrived, that the cerebellum was an organ of reinforcement to the spinal cord and other parts of the nervous system, has not been adopted and was even questioned by Mitchell himself in the light of later attention to cerebellar functions, but his paper nevertheless stands as a monument of original work, valuable for the actual data obtained and for the suggestions springing out of these. In a certain degree he anticipated Luciani's views as to the results of irritation of the cerebellum, and he clearly recognized the compensations by other parts of the nervous system for the losses in function the result of experiments causing deficit.

Mitchell is one of the few neurologists to whom well deserved fame has come because of his contributions to therapeutics. Foremost in this connection is of course that organized system of therapeusis usually spoken of as the Rest Treatment. One can scarcely realize at this time both the wide attention and the opposition which his advocacy of this treatment at first received. The value in cases properly selected of the physical measures included in this treatment,—seclusion, rest in bed, full feeding, with such adjuncts as massage, systematized movements, and electricity,—is now generally recognized, although the treatment was for a not inconsiderable time regarded by a large proportion of the profession not only as revolutionary but also by some as foolish or even harmful.

The psychic element in the treatment, which was fully understood by Mitchell himself, has not received due recognition, and some of the propagandists of modern psychoanalysis have denied all virtue to the treatment because of what seemed to them to be this lack. Mitchell was indeed one of the greatest of our psychoanalysts, although he was not given to prolonged, perplexing, and confusing methods of searching for doubtful causes in the sexual incidents of the early childhood or infancy of his patients. In this as in so many other matters of practical medicine, untrammeled by unnecessary and handicapping hypothesis, gifted with insight and confident in his own resources, he saw into the heart of nervous maladies. He was a sound exponent of the necessity of building the body while influencing the mind.

His first publication in book form of the rest treatment was in 1877 under the title of "Fat and Blood." This was preceded during several years by papers leading up to it on the milk diet and on rest in the treatment of special forms of disease. He had also given to the world in popular form his views in his brochure on "Wear and Tear."

In many minor but not unimportant ways Mitchell added to our knowledge of the use of drugs and other remedies, as in his observations and investigations on morphine and atropine, his recommendation of nitrite of amyl to abort epileptic attacks, his suggestion of lithium bromide as the most valuable of the bromine salts, his recommendation of the use of splints to procure local rest, and the employment of ice and sprays to reduce pain and relieve local spasm. He was among the first to promote the rational use of massage and faradic electricity, and he made numerous valuable suggestions as to surgical procedures like nerve section and nerve stretching, at a time when such suggestions had the merit of originality.

Time and again Mitchell directed the attention of neurologists to new clinical types, as to post-paralytic chorea in 1874, his description of this complex being the precursor in some respects of later published observations on cerebello-pedunculo-ruberal disease. Some of his important contributions were collected in his "Lectures on the Nervous Diseases of Women" in 1881, and his "Clinical Lessons on Nervous Diseases" in 1895.

The rare vasomotor neurosis, erythromelalgia or Mitchell's disease, was first fully described by him in a paper of unusual merit in 1878, although as early as 1872 he had called attention to the chief features of the disease, and continued its study in later papers.

With a mind always plastic and responsive, Mitchell, like some great men in pure science, Faraday for instance, when engaged in a physiological or clinical investigation, with every step taken saw new radiations of the subject in hand. One observation or discovery indicated to his alert mind other possibilities. This is illustrated in many of his studies which had a tendency to follow each other along expanding lines, as in those on the affects of accidental or surgical nerve section, on the psychic phenomena shown in cases of amputation, on the influence of barometric and other weather conditions in nerve injury and disease, and in his elaborate research with Morris Lewis on the physiology of the knee jerk and muscle jerk.

Mitchell's place as neurological investigator and writer cannot, however, be estimated by a mere enumeration of his contributions with brief glances at their main features. His work needs detailed consideration and full analysis. Brilliant suggestions and important deductions are often to be found in a sentence or paragraph.

The abilities of Mitchell as a psychoanalyst and student of nervous phenomena are shown in many of his delineations of character and recitals of incident in his works of fiction, as in his early novel, "In War Time," in "Characteristics," in "Dr. North and His Friends," in "Constance Truscott," in magazine articles involving such subjects as dual personality, and in much else that he wrote outside of his more formal and technical writings in the field of medicine.

Dr. Mitchell was not a promiscuous and over-frequent attendant upon medical societies, but he gave his attention to a chosen few, as the College of Physicians of Philadelphia, the Association of American Physicians, the Association of Physiologists, and the Philadelphia and national neurological societies. In the transactions of the College of Physicians of Philadelphia some of his best work, neurological and otherwise, is to be found. What he did for this great medical association and library will be told by others. To him more than to any other man it owes its present high position. As a member of the library committee of the college, with Dr. Dercum who was for many years associated with him on this committee, he was instrumental in adding many valuable neurological text books, treatises, monographs and journals, foreign and American, to the library collection.

Mitchell was one of the thirty-five original members of the American Neurological Association. The letter calling together the initial meeting for the formation of this association was dated December 15, 1874, and was signed by William A. Hammond, Roberts Bartholow, Meredith Clymer, J. S. Jewell, E. C.

Seguin, James J. Putnam and T. M. B. Cross. In this letter it was proposed to hold the first meeting on June 2, 1875, in New York city. Dr. Mitchell was one of the twenty-eight men to whom this letter was sent and as he accepted he became one of the original members. He did not attend the first meeting, June 2–4, but the committee on nominations named him for the office of president. He declined the honor and the first vice-president, Dr. Jewell, was elected to the office of president. In 1895, having been an active member of the association for twenty years, Mitchell was proposed and later unanimously elected honorary member. He was elected president of the association for the year beginning May, 1908. He presided at the annual meeting and read an address of much interest chiefly concerned with the discussion of neural energy. He was then in his eightieth year.

The writer with several colleagues having made plans for the organization of the Philadelphia Neurological Society, Mitchell on January 28, 1884 was chosen the first president. He held this position continuously from 1884 to 1890, constantly promoting the proceedings of the society by his presence, his work and his suggestions.

From time to time during the entire history of the society he contributed papers and presented or reported cases of much interest and value, drawing his material chiefly from his large private practice and the public service of the Orthopedic Hospital and Infirmary for Nervous Disease. It was at this hospital, which was at the time opposite the buildings of the University of Pennsylvania on Ninth Street, that I got my first glimpse of Mitchell working with his assistants on the study of patients coming to the out-door service. Even as an undergraduate student, I was impressed by his remarkable personality and his skillful clinical methods. This hospital and infirmary was established in October, 1867, at 15 South Ninth Street, in rooms over the shop of Kolbe, a dealer in medical instruments. Its neurological department designated as the Infirmary for Nervous Disease was opened in 1870 with Mitchell in charge. The hospital was removed to its present location, the northwest corner of Seventeenth and Summer Streets, in 1872. Mitchell continued his connection with the infirmary as attending physician and consultant until the time of his death. Here he gathered around him as years progressed able coadjutors and assistants. Some of his best papers written alone or in collaboration had their origin in the rich out-door and in-door services of this justly famous institution. Here he gave many of his delightful conversational clinics and conferences, some of which I had the pleasure of attending and out of which sprang the valuable collection of neurological essays included in his "Clinical Lessons."

In 1884, Mitchell served with Dr. H. C. Wood and the writer as consultant to the Insane Department of the Philadelphia Hospital, and although this service was brief, he did much in the way of pointing out the shortcomings of the institution at the same time indicating the best methods of remedying these and of improving the condition of the unfortunate insane who, then as now, were too much crowded for their comfort and scientific treatment.

Mitchell was instrumental in founding a ward for nervous diseases in the Presbyterian Hospital of Philadelphia, he acting as consultant and Dr. John K. Mitchell taking charge. He did much towards maintaining this ward besides giving his invaluable service as a neurologist. At the time of the foundation of the hospital he suggested the pavilion form of construction, knowing of its advantages from his Civil War experience. For some years, in the seventies, he was physician to this hospital.

When the opportunity came to me in 1877 to start a department for nervous diseases in the great charity hospital of Philadelphia and numerous obstacles were thrown in my way, I received from Dr. Mitchell, whom I consulted regarding the matter, such support and encouragement that I went forward with the project. He had before me seen the great value of the neurological material which was going to waste in the Philadelphia Hospital, but had not been able to accomplish his wish to turn this material to good use. He always took great interest in the clinico-pathological studies in cerebral localization and all forms of neurological work at this hospital.

As a trustee of the University and as one always interested in everything which tended to advance neurological science and practice, Mitchell gave his continuous sympathy to the development of the department of neurology in the University of Pennsylvania. The improvement and expansion of the hospital service, the work of the laboratory of neuropathology, the collaboration between the neurological and surgical departments, and the publication of annual and biennial volumes of Contributions all received his hearty endorsement and encouragement.

Mitchell was offered more than one professorial position. He sometimes told the story that he was professor in the University for a minute or two, having been elected while absent from the room by his colleagues of the trustees of the University of Pennsylvania to a chair which he resigned as soon as the fact was made known to him. For a short time he held a chair in the Philadelphia Polyclinic and did some post-graduate teaching in nervous diseases to Polyclinic and other students who sought his clinic at the Infirmary for Nervous Disease. The opportunities of this clinic were always hospitably given to the physicians of Philadelphia or to those visiting the city.

Unlike some men in science and medicine, Mitchell never feared that others might take advantage of what he knew or thought. He was generous in giving knowledge, and in affording assistance, this generosity showing itself not only in his willingness to help in matters of science, but extending also to giving personal help to those whom he believed needed and deserved such assistance. Just how much was owed to him by young physicians and especially those ambitious to pursue neurology can never be fully known. To those neurologists, who like myself are residents of the community in which Mitchell's long and useful life as a physician was largely spent, he was an ever present example and inspiration. Poor indeed was he in spirit who did not get something of value from contact with this master in medicine, this man among men.

TUBEROUS SCLEROSIS AND OTHER UNUSUAL FEATURES OF EPILEPSY[*]

N. S. YAWGER, M.D.
PHILADELPHIA

Although epilepsy is a most ancient disease, our knowledge of it in many respects is quite meager. And yet one of the greatest advances ever made in neurology was made with the establishment of the type of this disease which we know as jacksonian epilepsy. While the recognized manifestations of epilepsy are very numerous, there is still much of its etiology that remains a mystery. Of vast importance in the development of many cases is heredity, but the precise extent of the part this plays must always remain uncertain. In some instances we are confident that the cause is encephalitis dependent on the infectious diseases and sometimes it is due to traumatism, syphilis or brain tumor. Occasionally epilepsy is observed to follow fright or other mental shock, but the manner of such influence we cannot understand except as it acts on an individual who is a potential epileptic. Another interesting group of cases are those reported as exhibiting a variety of attacks, at times even said to be major, minor and psychic, which can in no wise be distinguished from those of epilepsy, but in which the attacks lessen or cease after the correction of some abnormal body condition as in intestinal intoxication. Since it is but seldom that an addition is made to our knowledge of the etiology of this disease, the matter of doing so seems deserving of more than passing notice. It now appears that tuberous sclerosis is the pathologic basis for a few cases of epilepsy.

For our first anatomic knowledge of tuberous sclerosis of the brain we are indebted to Bournville, and the appearance presented by the unhardened tissue is that of markedly indurated areas of sclerosis which are slightly elevated beyond the surrounding brain substance; in addition there are sometimes found small tumors protruding into the ventricles. Tuberous sclerosis in connection with glial formation may present the common picture of brain tumor.

---

[*] Read before the Section on Nervous and Mental Diseases at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

[*] From the Pennsylvania Epileptic Hospital and Colony Farm and the Department of Neurology of the University of Pennsylvania.

Recently Schuster[1] has made the clinical diagnosis in some of the feeble-minded and epileptic. Eight cases were reported, one of which came to necropsy. The identity of the disease in these cases he bases on the finding of adenoma sebaceum on the face, together with small angiomas, the latter appearing chiefly at the sides of the nose and on the chin; other cutaneous anomalies may be present. At times the internal organs, especially the heart and kidneys, are invaded by small growths which may give rise to some of the symptoms found in the chronic disorders of these organs. The skin manifestations may to a less extent be met in some of the blood relatives. One man was seen who showed typical adenoma sebaceum without any other appearance of disease, but he had a daughter of 6 years who was an epileptic imbecile. Schuster states that tuberous sclerosis must be interpreted as a conditionally, endogenous, hereditary family disease. He furthermore refers to a relationship with Recklinghausen's disease and says that while tuberous sclerosis involves chiefly the brain and skin, in neurofibromatosis it is for the most part the peripheral nerves that are implicated.

More recently, however, Nieuwenhuijse,[2] in studying the question of a relationship between these two diseases, writes that their clinical appearance and also their anatomic changes are entirely different, that the skin fibromas which occur in both affections have their own individuality, and that when the brain is involved in Recklinghausen's disease, there is found a series of absolutely typical changes which are in no wise like those found in tuberous sclerosis.

Sailer[3] in 1898 recorded the finding of hypertrophic nodular gliosis in the brain of an epileptic idiot, and Munson[4] in 1912 reported his findings in an infant of

---

[1] Schuster: Deutsch. Ztschr. f. Nervenheilk., 1913, p. 96.

[2] Nieuwenhuijse: Ztschr. f. d. ges. Neurol. u. Psych., orig., 1914, xxiv. 53.

[3] Sailer: Jour. Nerv. and Ment. Dis., 1898, p. 403.

[4] Munson: Jour. Nerv. and Ment. Dis., 1912, p. 251.

27 months who clinically showed only seizures and possible mental enfeeblement. At necropsy, he says, "Throughout the cerebrum are foci of hardening corresponding to single convolutions or to groups of convolutions. These are much harder than the intermediate cortex, and in places seem to be so hard as to be almost 'bony' to the touch." Munson, on anatomic grounds, placed the case with the true multiple scleroses. From the descriptions given by these writers I think it highly probable that both brains showed areas of tuberous sclerosis. I am not aware of any case that has been reported in English literature in which the clinical diagnosis of tuberous sclerosis has been made.

We have two patients at the Oakbourne Epileptic Colony which present the features described by Schuster in some of his cases. In neither of them has there been evidence of focal involvement of the brain. It is regretted that a complete family study has not been possible, but one patient is from a distance, both are of foreign-born parents who are very ignorant, and as a result our information in this respect is very fragmentary.

CASE 1.—The first patient is a girl of Italian descent, aged 14, whose attacks began at 7 years. She is one of six children, and the others, it is stated, are healthy. She was never robust and is said to have had rickets in infancy. Mentally, she is an epileptic imbecile and temperamentally she is morose and abnormally sensitive. The seizures are major in type, and three and one-half years ago she had status which lasted eleven days. Physically, her appearance is somewhat grotesque with a possible element of achondroplasia. There is also something suggestive of cretinism, and with this thought in mind we placed her on thyroid extract. For a time decided improvement was made and this was manifested in various ways, in six months she gained 15½ pounds in weight, there was an increase of 2 inches in height, she was less irritable and much more alert mentally, and while there were on an average of twenty-five seizures a month before the administration of thyroid, during this treatment for a period of six months she was free from attacks: unfortunately, she relapsed. This individual (Fig. 1) is small in stature, her legs are abnormally short and her hands reach almost to the knees; she is loose jointed, shows marked lordosis and has a protuberant abdomen; her features are thickened and the thyroid is not palpable. The pulse-rate is usually from 90 to 100 and the specific gravity of the urine is always low, sometimes not more than 1.002, but without albumin or casts. She is surprisingly tolerant of cold, never appearing uncomfortable even though thinly clad in the coldest weather.

The following very complete dermatologic report is by Dr. O. H. Foerster: On both cheeks, grouped above the nasolabial furrows, are numerous small pin-head to wheat-grain-sized papular tumors, discrete and confluent, firm, with convex summits, and of brown and yellow-red color. The larger growths are associated with well-marked telangiectases and on pressure are yellow-red in color. The remaining portions of both cheeks are clear, except for several similar isolated lesions of half-pin-head size. On the chin are about twenty similar discrete lesions of less than pin-head size.

Both alae nasi, and tip and dorsum of nose are practically covered by waxy or translucent, pink to yellowish, elevated, convex, closely set and confluent, papular growths of firm consistency. Several are associated with telangiectases, though less developed than on the cheeks. Similar, more rudimentary lesions of less than one-quarter pin-head size, and yellowish in color, are situated on the upper lip. In both nasolabial angles the lesions are more prominent and grouped, so as to give a verrucous appearance. On the left upper eyelid are several tiny lesions with color of normal skin.

On both sides of the forehead, extending toward the eyebrows from just within the hair-margin, are two elevated tumor masses of irregular outline, light yellow-red in color, with smooth flat surface except for several shallow clefts or furrows. The entire right temporal region down to a line between the outer edge of the right eyebrow and the right ear is occupied by a firm growth with irregular margins, of yellow-red color, lobulated on deep palpation, and of verrucose appearance. Embedded in the scalp at the right parietal region and at the occipital protuberance are two firm, ovoid, elevated masses of finger-nail size. On the neck are several small dark, pedunculated fibromas.

The middle third of the forehead is studded with comedones. The tongue, buccal and labial mucous membranes are normal. The lobes of both ears are abnormally large.

The extensor surface of the arms and dorsum of hands, from knuckles to above the elbow, is of light brown color, sprinkled plentifully with pin-head and slightly larger non-pigmented areas, with irregular, white patches of finger-nail size at the elbows and on the radial side of the right wrist. On the dorsum of the right wrist, on the radial and ulnar side, are two irregular, scar-like, slightly elevated lesions, with a "knitted" appearance, and spots of light-yellow to brown pigmentation. Palms and finger-nails are normal; hair-growth is normal.

Both legs are darkly pigmented, especially at the knees, and show several areas of entire loss of pigment, from finger-nail to small palm in size.

The skin of the back is of darker color than normal, with a moderate eruption of acne on the shoulders, upper back and in the sacral region. On the right shoulder is a group of three fibromatous yellow-red growths. Arranged irregularly in a belt-like manner just above the hips are several flat fibromas.

Case 2.—The second patient is a girl of Slavish parentage, aged 15. It is said that her father has some curious markings on his face and that he has periodic headache and fainting-spells. A paternal uncle also has attacks of headache. At 3 years this girl was hit on the head by a ball and was unconscious for a time. Her attacks began at 4 years and her rank is that of an epileptic imbecile. For a time the seizures occurred daily but gradually they became less and less frequent and now she is for months without attacks. This individual presents several unusual features. Her voice is peculiar, sometimes it is feminine in pitch, at others masculine and again it will "break" as is common with the male voice at puberty. Though she had many major attacks previous to her admission to the colony, during her stay of two and one-half years with us, she has had only three complete epileptic seizures. Twice there have been vasomotor discharges in the form of intense redness which has covered all exposed parts of the body. Ten paroxysms of uncontrollable laughter have been recorded and three of these have occurred during sleep. There is a cartilaginous thickening of the left ear which has probably resulted from a hematoma auris. The palatal arch is high and narrow. This patient (Fig. 2) is markedly stoop-shouldered, she carries her head far forward and has a decidedly ungainly gait.

Dermatologic report by Dr. Foerster: In the flush area of the face, arranged with considerable symmetry, and grouped especially in the region of the nasolabial furrows and on the chin, are numerous pin-head and slightly larger tumors, many with telangiectases on their summits. They resemble in every particular the lesions described in Case 1, but are in general more rudimentary, though equally characteristic. Similar pigmented patches and flat nodular fibromas are present, though fewer in number and smaller. In addition there is a large flat nevus of the size of an adult's palm to the right of the lumbar spine.

SOME UNUSUAL ETIOLOGIC FACTORS

A remarkable cause of epilepsy is mentioned by Oppenheim[5] as having been observed by Marburg. It occurred in an individual after eating for many years from 1 to 1⅓ ounces of roasted coffee-beans a day. I have recently examined a patient in whom the exciting cause was most unusual. A man aged 20 of good heredity and correct habits had his first seizure immediately after a drive of several hours' exposure to a temperature of 20 degrees below zero. He now has attacks every month or two and is an undoubted epileptic. What change was brought about by this exposure we do not know, but perhaps it only aroused a slumbering epilepsy. The disease sometimes follows an electric shock, and Jolly[6] has recently recorded the instance of a motorman 20 years of age and previously healthy who on climbing to the top of his car came in contact with the charged pole; he was rendered unconscious. The next day he had pain in his head and fourteen days later there was an unconscious attack; after this there appeared complete epileptic seizures. It is admitted that the electric current may have been only the exciting cause. Spratling[7] speaks of a man of 32 years who had never had convulsions but who became an epileptic after receiving an electric shock. It is not stated that he was rendered unconscious by this shock.

---

[5] Oppenheim: Lehrbuch Nervenkrankheiten, 1913, p. 1590.

In a patient recently seen there must have been some actual destruction of cerebral tissue. It was a patient of Dr. Henry Pleasant's, aged 26, and an electric worker; he came in contact with an alternating street current and was rendered unconscious. Later it was observed that he had a hemiparesis involving the right face, arm and leg. Following this was severe headache and at a time impossible to determine definitely, he began with minor attacks and three years after the shock he was having complete epileptic seizures.

Severe injuries from electricity usually cause death, and from the non-serious ones a good recovery may generally be expected. The changes most frequently observed in the brains of criminals who have been electrocuted are capillary hemorrhages, arterial anemia and venous congestion; more or less destruction of nerve-cells has been reported by one writer.

Gowers[8] speaks of the influence of light as exciting seizures in rare instances. He refers to two cases, one a girl who had just recovered from typhoid and whose first fit occurred immediately on going into the bright sunshine. At no other time did she have attacks. The other was a man whose attacks were always brought on by looking at a bright fire. Gowers states that the discharge apparently began in the visual center. A somewhat similar effect is seen in one of our cases. It is that of a young man whose face and neck muscles are thrown into most vigorous clonic contractions whenever a bright light falls on his eyes. At times the head is thrown backward violently and it may be held there as much as thirty seconds. Rarely, these clonic movements pass into complete epileptic seizures, but nearly all of his major attacks appear precipitately. Very rarely there is spontaneous recovery from epilepsy. The following case is one in which all motor manifestations ceased but a paraplegia persisted: A boy at 2 years developed fever, was nauseated, had general convulsions and then the right arm and leg became helpless. He probably had an attack of acute poliencephalitis. The right arm and leg remained spastic, and jacksonian convulsions were manifested from time to time until 13 years, when the attacks ceased spontaneously. They stopped, strange to say, at an age when epilepsy is very prone to begin. He has now been for eleven years without attacks.

---

[6] Jolly: München. med. Wchnschr., 1912, p. 1453.

[7] Spratling: Epilepsy and Its Treatment, 1904, p. 124.

[8] Gowers: Epilepsy and Other Chronic Convulsive Disorders, 1885, p. 75.

The next case is one in which jacksonian convulsions developed into or at least were masked by complete epileptic seizures. The patient is rather remarkable because of the great variety of nervous phenomena displayed. This girl probably also had an attack of acute poliencephalitis at 2 years. In the month of May she had fever and convulsions and lay unconscious for a week. On regaining consciousness it was observed that there was disability of the right face, arm and leg. For three weeks the child seemed aphasic; at first she did not talk at all but soon learned again. From time to time slight motor disturbance occurred on the right side and at 10 years the child was placed in a home for crippled children. When she began to use her pen, which she held in her left hand, she wrote mirror-writing; it was with difficulty that the child learned to write correctly. By this time there were pronounced jacksonian seizures and at 14 years attacks developed in which the convulsions were general and the unconsciousness absolute. The patient's acute illness in infancy was followed by aphasia, right hemiplegia, jacksonian convulsions which developed into complete epilepsy, and she also exhibited athetosis, associated movements and mirror-writing.

Another patient is an epileptic girl with myoclonus and recurring sighing manifestations. Her age is 13 and the attacks began at 3 years without obvious cause. The seizures are both major and minor. There is often observed a distinct bodily movement in an arm, a leg or the head and sometimes the shoulders are shrugged. In addition there are less-marked contractions of the muscles in the face and in other parts of the body. All of these movements may be observed during sleep. The patient often sighs deeply both when asleep and when awake. There are at times diaphragmatic spasms. The myoclonus is always more marked after seizures. There are likewise lesser movements that may be felt but not seen. At all times by grasping an arm or leg, a distinct muscular contraction may be felt within the part, and the feeling transmitted is that of throbbing or pulsation.

SLEEP AND EPILEPSY

The relation between sleep and epilepsy is most curious and interesting. Most epileptics after a complete attack have a temporary awakening and from this they usually drop into deep sleep. Rarely, this sleep is absent as shown in the following case: A girl aged 18 had her first attack two days after the burial of her mother. She is now 33 years old and has many short but severe attacks. She injured herself so often and so badly that for a long time a protector was worn on her head. The seizures are both nocturnal and diurnal, but now for some months all attacks have been confined to the periods of sleep. This patient has never slept after an attack and even though the seizure occurs during the deepest sleep, she invariably awakens at once and remains awake from a half to two or three hours.

In some individuals sleep is the only known cause of recurring attacks. It is said that in most epileptics the seizures have begun during sleep, that often it is in the deepest sleep that a paroxysm occurs and that more attacks take place when asleep than when awake.

There are momentary losses or absences which have the appearance of sleep, but they are something more than this, for attacks of some kind are frequently seen while these individuals are actually asleep. Besides, if stuck sharply with a pin while in such attacks, unconsciousness as a rule is uninfluenced; any other person at such a time would awaken at once. Such seizures resemble more nearly the disturbance of consciousness seen in cardiac syncope. In connection with this subject, I have observed a very curious case. It was a girl of 19 years whose attacks began at 10 years without known cause. She has major and minor epilepsy with seizures occurring eight or ten times a day. She also has hallucinations of sight and these usually appear just before her attacks. This patient is very drowsy and if permitted, would sleep as much as fifteen hours out of twenty-four. She always takes several naps during the day and will even fall asleep while scrubbing the floor. At one period of about a month's duration, she appeared to be passing into sleep incessantly; all day long she would begin

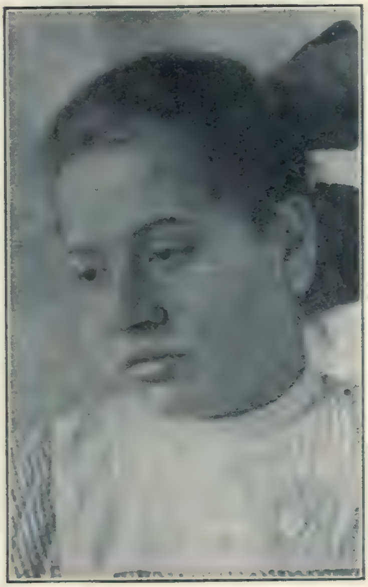


Fig. 1.—Epileptic imbecile showing sebaceum, angiomas and tumor masses.

to doze and then promptly recover herself; and this happened several hundred times a day for several days. Considering her drowsy condition, it is difficult to know where to place this phenomenon. Was the manifestation psychic attacks of epilepsy, was it narcolepsy or was this individual an epileptic exhibiting attacks of narcolepsy?

SO-CALLED NON-EPILEPTIC "ABSENCES"

Friedmann[9] asserted that certain cases previously classified as showing petit mal were really instances of non-epileptic "absences," and furthermore stated that such seizures probably had no relation to hysteria. He described fifteen personally observed cases and regarded the seizures as narcoleptic in nature. This subject has recently been referred to in this country by Sedgwick.[10] In this connection Gowers[11] states that

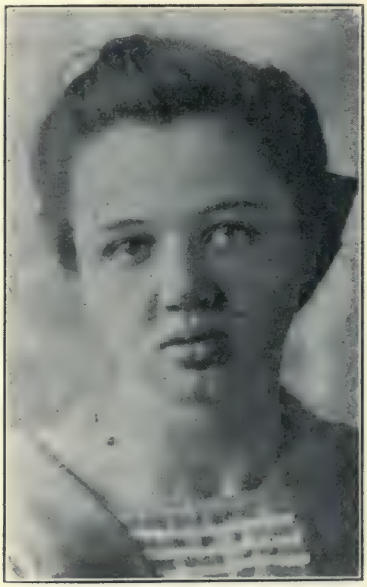


Fig. 2.—Epileptic imbecile showing the same condition as in Figure 1, though more rudimentary.

---

[9] Friedmann: Deutsch. Ztschr. f. Nervenheilk., 1906, p. 462.

[10] Sedgwick, J. P.: Spasmophilia with Especial Reference to Familial Reactions and Repeated Absences, Am. Jour. Dis. Child., February, 1914, p. 140.

[11] Gowers: Borderland of Epilepsy, 1907, p. 116.

"before fifteen cases of narcolepsy were met, 30,000 cases of epilepsy would come under observation! Most of these cases were probably minor epilepsy." The features of this disorder are, according to Friedmann. attacks which usually begin between the ages of 4 and 7 years in apparently healthy children; they consist of a momentary disturbance of consciousness and almost without convulsions or falling; they recur from six to ten and up to 100 times a day; it is only occasionally that they appear at night and frequently they are recovered from; the physical and mental development of the child is not affected.

I have studied two such cases. The first is a girl aged 14 whose attacks began at 9 years without obvious cause. The seizures vary in number from six to thirty per day and are of from ten to fifteen seconds' duration. This momentary disturbance of consciousness is not preceded by an aura; never has there been a convulsion. This girl is healthy, growing rapidly, unusually active and has the mentality of a normal child. So far she conforms to the description of Friedmann's cases. But on further careful study, many attacks are seen while she is asleep; she was once known to fall and in a number of attacks has wet her clothes. If sitting and caught at the time of a seizure her arms and legs are found to be slightly rigid; but if walking she continues though sometimes with slight wavering; occasionally, the movements of tasting are seen. After some of the seizures there is an action suggestive of straightening out the clothes, and this we know is common among epileptics.

The second patient is a girl of 16 years whose so-called "absences" began at 6 years without obvious cause. The attacks usually vary from six to ten per day but they have gone as high as 100. The usual duration is from fifteen to twenty seconds. There is no aura and while she is generally unconscious, some seizures are so light that they are barely apparent even while one is watching her. She also is healthy, is growing rapidly and is of average mentality though somewhat peculiar. During the nine and one-half years of the disease there have been two convulsive attacks with complete loss of consciousness. If spoken to while in a spell there is no response; if stuck sharply with a pin at the beginning of an attack, the seizure is aborted; but if fairly under way the hand is drawn from the irritation although she has no subsequent knowledge of having been stuck. There is an occasional drowsy episode during which time the patient can hardly be kept awake. Since the age of 14 a very interesting feature of her clinical history is recurring attacks of complete migraine; these usually appear three or four times a month. The intelligence of this girl has made her description of the attacks interesting. The migraine is preceded by the appearance of a bright light before one or the other eye but never before both in the same attack. This light is shaped like the flame of a gas-jet with a serrated upper edge and an attenuated extremity which always points to the outer side and which the patient speaks of as the "tail." The light lasts about twenty minutes and before it disappears headache begins; this is always preceded for about a minute by double vision. The headache usually persists for a half hour and frequently there is nausea and vomiting. When the attack subsides she feels perfectly well except for extreme weakness.

I believe the "absences" in these two cases are best classified as momentary psychic attacks of epilepsy. It may be that Friedmann and others have regarded the motor manifestation as the cardinal symptom in epilepsy, whereas the disturbance of consciousness is now generally accorded first place.

It is obvious that a great variety of momentary phenomena important in the matter of diagnosis may be lost unless the patients are constantly under the care of trained and observant attendants.

To me, the conclusion that these two patients are epileptics is inevitable.

The last patient has associated with her epilepsy attacks of migraine. As to the relation of these a wide difference of opinion exists; but some light should be thrown on this question by a careful study of the cases in which there is a clinical history of both affections.

2005 Chestnut Street.

Reprinted from The Journal of the American Medical Association
Oct. 17, 1914, Vol. LXIII, pp. 1348-1352

Copyright, 1914
American Medical Association, 535 N. Dearborn St., Chicago

ACUTE MYELITIS, SECONDARY TO PERIRECTAL ABSCESS, DEVELOPING A FEW HOURS AFTER SEVERE FRIGHT *

WILLIAM G. SPILLER, M.D.
Professor of Neuropathology, University of Pennsylvania, Department of Medicine
PHILADELPHIA

Fright has been considered, especially by German authors, a cause of organic paralysis, and cases with this etiology are reported in the older literature. It is probable that many of these cases were instances of functional paralysis and not organic. In 1903 I had the opportunity of studying a case in which a struggle with burglars was followed in two days by paresthesia of the lower limbs of sudden onset and some days later by motor and sensory paralysis of these limbs and then death. Pus within the abdomen extending from the rectum and severe myelitis were found. The fright may have had no connection with the paralysis, but on the other hand it may have disturbed the circulation in the spinal cord and in connection with the exertion caused by the struggle have rendered the cord more liable to infection.

H. E. S., white, first came to the nervous dispensary of the University Hospital, Nov. 9, 1900. He was a railroad conductor, aged at that time 35 years. While coming from Omaha he had become paralyzed in the right side of his face after he had been sitting by an open window. The paralysis was severe, and the entire right side of the face was affected. He made a complete recovery from this paralysis.

April 22, 1903, he returned to the nervous dispensary, when the following notes were taken: He had been perfectly well since he had had the facial palsy. He denied venereal disease. He had been married five years, from 1891 to 1896, but his wife had never been pregnant.

---

* Read before the Section on Nervous and Mental Diseases at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

During the night of April 17, 1903, he was attacked while in an eating-house. He was the proprietor of this restaurant and at 1 o'clock in the morning three men entered his place of business and tried to rob him. With the assistance of another man who was in the place, he succeeded in driving off his assailants and it is very certain that he was not injured bodily in any way, although he was very much excited.

During the afternoon of April 19 he was sitting by a window in his house. He had been perfectly well until this time. While he was sitting by the window the man who had assisted him in expelling the robbers passed and the patient's thoughts were directed to the attempted robbery. Soon after the man passed he rose from his chair but noticed that both of his legs were asleep, as he expressed it, and he walked therefore with some difficulty. The paresthesia has persisted until the present time. When he first experienced the paresthesia his feet and right leg seemed numb, and gradually the numbness spread to the knee on the left side and to the hip on the right side, implicating also the penis, scrotum and gluteal region. The rest of his body seemed normal. He knows when he has passed urine or when the rectum should be emptied, but he cannot feel the passage of the urine or feces. He has not had involuntary micturition or defecation. He has not had any sexual desire or seminal emission since his symptoms first appeared, but the time has been short. He tires very readily and needs to make more effort to perform voluntary movements. The station is fair even with eyes closed.

Standing on the right leg alone is not so easy as standing on the left leg alone. He moves all the joints normally. He can place his heel at any desired point with fair accuracy. Objective sensation in its different forms is little if at all affected; when tested he occasionally gives an incorrect answer, but soon corrects himself. He complains that he feels as though he were walking on felt and that he does not seem to have shoes on.

Both patellar and Achilles tendor reflexes are normal. In testing the Babinski reflex the large toe seems to go up first and then to go down. The reactions of the irides are normal.

April 27: The Babinski reflex is positive on each side.

May 2: Since Sunday, April 19, the patient had been able to walk without difficulty and came to the dispensary, although his gait was slightly ataxic. The toes of each foot had been forcibly extended from irritation of the sole of the foot. He had no marked disturbance of gait until Thursday morning, April 30, when, about 11 a. m., he fell to the floor but was able to pull himself on the sofa. He had not passed urine since half past five of that morning. So soon as he climbed on the sofa he began to have pain in the lower part of the abdomen. Dr. Lindauer was sent for and catheterized the patient. The man had not had any pains in the lower limbs at any time although he had pain in the lumbar region. Bowels have not moved since April 30.

About 2 p. m., April 30, the patient succeeded in rising from the sofa and by supporting himself was able to pass into another room on the same floor. He sat in a chair three-quarters of an hour. Later he walked into the front room and walked several times about the room until he was brought to the University Hospital about 6 p. m., April 30.

Friday, May 1, the morning after admission to the hospital, he was able to stand and he walked around the bed. Dr. Nisbet, about 3 p. m. of this day, had the patient get out of bed and stand by the side of the bed, supporting himself slightly with one hand. As he was unable to void urine while standing, he was catheterized.

Resistance to passive movements in the lower limbs was nearly normal, and he could flex and extend the leg and thigh even when these movements were resisted. The patellar reflex was exaggerated, especially on the right side. Ankle-clonus and Babinski reflex were distinct and both were more marked on the right side. Tenderness to pressure over the vertebrae was not felt.

Condition May 2: The lower limbs are not at all atrophied but are completely paralyzed, so that there is scarcely any movement in the toes or hips. The patellar reflex on each side is not so prompt as it was yesterday; it is about normal on the left side and slightly below normal on the right side. Ankle-clonus is persistent and typical on each side, but more pronounced on the right side. Babinski's reflex is very distinct on each side; on the right side the movement of the big toe upward is typical and slow; on the left side the movement of the big toe upward is of the same quick character as it was last week. The lower limbs are cool to the touch and are spastic.

Tactile sensation is much impaired in the lower limbs, beginning about 2 or 3 inches below a line passing through the umbilicus. The outer part of the right thigh and the feet are almost anesthetic. Pain sensation is acute in the lower limbs except on the outer side of the right thigh, but is not so acute as in the upper part of the trunk. Sensation for temperature is much impaired in the lower limbs and over the abdomen below the umbilicus, but the sensation seems to vary during the examination. The abdomen is very rigid and painful to slight touch.

The movements of the upper limbs are free and the motor-power is normal. The grasp of each hand is normal. Sensation for touch, pain and temperature in the upper limbs and face is normal.

The reaction of the iris to light and in convergence is prompt in each eye. The movements of the eyeballs are normal in all directions. The tongue is protruded in the median line. The muscles of the facial and motor trigeminus supply are normal.

May 3, 2 p. m.: The lower limbs are completely paralyzed. The patellar reflex is absent on each side even on reinforcement. Babinski's reflex is present on each side. Ankle-clonus is not obtained on either side. The abdomen is rigid and tender. Sensation for touch in the right lower limb is lost on the outer side below the crest of the ilium, on the anterior side below Poupart's ligament, on the inner side below the patella, and on the posterior side below the popliteal space. In the left lower limb tactile sensation is impaired below the knee. Pain sensation in the right lower limb is lost on the outer side below the trochanter, and on the inner and outer surfaces of the leg below the patella, and is impaired in the rest of the right lower limb. It is lost in the outer part of the left leg 2 inches below the patella to 2 inches above the ankle.

Heart and lungs are normal.

May 5: The upper limbs are not at all affected. Respiration is entirely costal; the abdomen is distended. Since yesterday morning the patient has been having intense pain over the trunk back and front from about 4 inches below the nipples to the inguinal region. The whole spinal column from below the spines of the scapulae is very tender to pressure. Sensation for touch and pain is entirely lost in the lower limbs and abdomen as high as a line about 2 inches below the umbilicus. Patellar reflex and Achilles tendon reflex are completely lost on each side. Babinski's reflex is still very distinct on each side. The man has frontal headache. He complained of a sensation of fulness in the bladder which was removed by catheterization.

Death occurred May 9, 1903.

The necropsy was performed by Dr. William S. Wadsworth, coroner's physician. He reported that the findings showed such clear evidence of gradual extension from the region of the rectum that he gave as cause septicemia by extension of infection from the rectum. Dingy greenish pus was found in the tissues about the rectum with emphysema and cellulitis extending to the buttocks above the brim of the pelvis, implicating the retroperitoneal space and the lymph-glands of the abdomen, extending as high as the pleura, following the nerves to the spinal cord, and causing general peritonitis. There was evidence of beginning pleurisy. The bladder did not contain pus.

Dr. J. S. Evans, Jr., reported, May 16, 1903, that intradural cultures were negative, subcutaneous cultures were contaminated, but a culture taken the day before death from the area of cellulitis of the buttocks showed the presence of the *Streptococcus pyogenes.*

My findings in the spinal cord were as follows: Lumbar Region: A section from the midlumbar region showed much softening although it was possible to distinguish white from gray matter. The anterior and posterior roots stained deeply by Weigert's hematoxylin method. The nerve-cells of the anterior horns were much diseased; by the thionin stain, they contained few chromophilic elements, and had very imperfect dendritic processes. The nucleolus stained deeply and it was a little difficult to make out the nuclear membrane. In some of the cells of the anterior horns the chromophilic elements had entirely disappeared. There were recent hemorrhages at some places in the cord, but no accumulation of pus-cells could be found in the cord. Sections from the lower thoracic region showed more disintegration than did those from the lumbar region, and it was impossible to distinguish here white from gray matter. Collections of round cells were seen within the cord.

There was diffuse staining with the Marchi method in the lumbar region and to some extent in the thoracic region, but it was not intense.

The nerve-cells of the anterior horns in the cervical region were much diseased. They stained deeply, were shriveled, had few processes, and in some the nucleus was peripheral. The anterior and posterior roots were normal in the cervical region.

The fright in this case was very pronounced. Could it have had any relation to the paralysis?

The tendency is to attribute all acute paralyses developing immediately after fright to hysteria, and this tendency is justifiable in most cases; but there is a possibility that fright may act as an *agent provocateur,* in the sense of French writers, and be a cause of organic change. Papers bearing on this subject are found chiefly in the older literature. In many instances the fright is only a coincidence, but in others it may affect the circulation in vessels already diseased. When syphilitic endarteritis has occurred in the vessels of the cord, apoplectiform myelitis has been observed not infrequently. I have already written on this subject.[1] Paralysis under such circumstances may develop within half an hour without any apparent exciting cause; how much more probable is paralysis, therefore, when severe emotion disturbs an impaired circulation. Blushing shows chiefly in the face and may be intense as a result of mild emotion, but the blushing is not confined to the face and may affect a large part of the body. Are the interior organs implicated in blushing? It is at least possible. A congestion of the vessels of the spinal cord from vasomotor disturbance when those vessels are diseased may cause thrombosis, and this certainly may cause paralysis, or, if the thrombosis be mild, the vitality of the affected part of the cord may be reduced and the susceptibility to toxins may be increased. One may well hesitate at the present time to accept as proved acute organic paralysis as caused by fright in cases in which the vessels are in normal condition. It may be well to review briefly some of this older literature on fright paralysis.

---

[1] Spiller, W. G.: Jour. Nerv. and Ment. Dis., October, 1909.

Kohts[2] refers to fright as a cause of exophthalmic goiter, icterus, turning white of the hair, and cerebral apoplexy. He had an unusual experience in that he was able to observe a great many cases of nervous and other disease that developed during the severe bombardment of Strassburg. During the thirty-one days of the siege the town was bombarded almost every night; 193,722 bombs were thrown into the city, and after the surrender only two houses showed no marks of the firing. Kohts reports cases of paralysis agitans, and of myelitis and other diseases which developed immediately after severe fright, one of which was with necropsy. The people of the city took refuge in the cellars, and repeatedly saw those about them killed. The effect of psychic trauma has probably never been more clearly shown. Kohts offers no explanation for the manner in which fright leads to organic disease. It is difficult to attribute such an array of cases to mere coincidence.

Since the investigation of emotional paralysis by von Leyden and Kohts, very little attention has been given to the subject, although their publications first appeared in 1873 and 1874.

The case of Todd,[3] described by him as emotional paralysis, is questionable as regards an organic basis, according to von Leyden.[4] Hine's[5] case was one of chorea of pregnancy developing after severe fright. The midthoracic region of the cord was softened.

---

[2] Kohts: Berl. klin. Wchnschr., 1873.

[3] Todd: Clinical Lecture by Beale, London, 1861.

[4] Von Leyden: Klinik der Rückenmarks-Krankheiten, 1874, i, 173; 1875, ii, 181, 190, 430; Ztschr. f. klin. Med., 1880, i, 12; Berl. klin. Wchnschr., Feb. 20, 1905, p. 193.

[5] Hine: Med. Times and Gazette, 1865, cited by von Leyden.

Pasque's[6] case was one of paralysis of the lower limbs cured by strychnin.

Leyden reported the case of a woman aged 21 who was awakened by fire in a house opposite the one in which she was. She was greatly frightened. Immediately after the fire was extinguished and she had returned to her bed, she experienced severe pain in the left leg. The following morning the left leg was weak and gradually spastic paraplegia developed. A diagnosis of thoracic myelitis from fright was made. The necropsy showed sclerosis in the midthoracic region of the cord.

The second case that Leyden reports was as follows: A girl aged 23, previously healthy, during the siege of Strassburg found on returning home that her house was on fire. Symptoms began immediately and gradually the lower limbs became paralyzed and contracted. The necropsy showed chronic diffuse myelitis with meningitis, the upper thoracic region was most affected.

Another case reported by Leyden is as follows:

A servant girl, aged 16, was greatly frightened by an attempt to rob a house. The two nights immediately following the attempted robbery she slept badly. On the morning of the third day following the psychic trauma, or more exactly, after sixty hours, the lower limbs became weak. Paresthesia soon appeared in both upper limbs as well as pain in both lower limbs, and the patient became unable to move any of her limbs and had flaccid paralysis with complete loss of sensation in all the limbs. She had retention of urine and incontinence of feces only during a few days, and the disturbance of the trunk disappeared, so that motion and sensation were lost except in the head, neck and trunk. She had no mental symptoms. Tendon reflexes became normal, and Babinski's reflex and electrical reactions were normal. The case described above Leyden regarded as functional, and the result of suggestive treatment demonstrated the correctness of this view.

Brieger[7] says that organic cases caused by fright or mental shock have been reported by Todd, Hine, Pasque and Berger, but all were without anatomic foundation. Leyden was the first to show that such a cause might produce tissue changes in the central nervous system. He refers to the case in which Leyden found sclerosis in the midthoracic region, and to another in which he found diffuse sclerosis of the upper and middle thoracic regions. Brieger knew of no other anatomic evidence at the time he wrote his paper. His patient was a prostitute, aged 23, who had the symptoms of lumbothoracic myelitis developing first within a few hours after she was much frightened by a drunken man. The necropsy revealed a myelitis in the upper lumbar and lower thoracic regions. The patient had had syphilis. Brieger says clearly that fright was the only cause of the myelitis. It was not syphilitic, because no signs of visceral syphilis were found, but the syphilitic infection and the irregular life had made her more vulnerable. Still one may suspect syphilis notwithstanding Brieger's opinion. He thinks that the fright may have caused a temporary interruption of the circulation in the spinal cord, the lumbar region of which was exhausted by recent prolonged dancing. Fright affects the stomach and may cause vomiting, loss of appetite, ulcer and hematemesis, and may affect the lumbar region, causing pollution, etc. It is questionable whether the explanation given by Brieger can be accepted. The necessity of frequent micturition preceding examinations is well known by students.

---

[6] Pasque: Arch. de méd. belge., December, 1842.

[7] Brieger: Ztschr. f. klin. Med., 1881, ii, 121.

P. K. Pel,[8] writing in 1881, in describing this *Schrecklähmung,* said that while functional disturbance following fright is well known, other cases are observed in which disease of the spinal cord has followed fright. Such are the cases of Russel-Reynolds, Todd, Hine, Leyden, Kohts, Brieger and others. Such cases are rare. Pel's case seems to have been one of functional paralysis following fright.

A case reported by Daus[9] is a valuable contribution to fright paralysis. A boy, aged 14, was physically poorly developed, and was obliged to walk much, but was healthy until Nov. 16, 1901. On this day he was so greatly frightened by an automobile that without being struck by it he fell to the ground and did not lose consciousness. When he tried to rise he was unable to do so, and he was carried home and put to bed. He had severe pain in the feet and both lower limbs were anesthetic as high as the gluteal region, and he had rectal and vesical symptoms.

---

[9] Daus: Monatsschr. f. Psychiat. u. Neurol., 1903, xiv, 66.

[8] Pel, P. K.: Berl. klin. Wchnschr., June 6, 1881, p. 325.

Daus says that the most commonly affected region in fright paralysis is the lumbar cord, and this is because of functional overuse of this region. In his case there was no evidence of sexual abuse, which I have known to be the immediate cause of paralysis from lesion of the spinal cord, but the boy had been much on his feet, and in this way may have rendered his lumbar region more susceptible, but one would suspect some undiscovered cause in addition to fright. His case seems to show that the epiconus region has especial vulnerability to *Schrecklähmung*.

I have known persistent paralysis of the lower limbs to develop rapidly in a man during the early morning following repeated sexual intercourse occurring the preceding night.

The literature that Daus refers to on *Schrecklähmung* is of ancient date. With the exception of Leyden's paper in 1905 and Daus' in 1903, the subject seems to have been ignored by almost all of the recent writers.

Emotion has seemed in some cases to be the cause of hemiplegia in a person previously healthy. Cases of this kind have been oberved by Babinski[10] and Oppenheim,[11] and another was reported by E. Frank.[12] A woman, aged 26, developed left hemiplegia after a severe fright. The paralysis was believed to be organic and the result of a pontine lesion. No cause for this hemiplegia could be determined; the heart appeared to be normal, and at first no signs of syphilis could be detected, but the Wassermann reaction was positive. Careful examination revealed small scars near the mouth, the result of a facial eruption at the age of 5 years. The history of corneal opacity at the tenth year was obtained. Hereditary syphilis seemed probable. It is not likely that a healthy vessel would rupture from an emotion, but a diseased pontine vessel might yield, and the blood leak gradually.

Urinary paraplegia has been discussed in the older literature, but now one hears comparatively little of it. The paper of T. J. Walker has attracted considerable attention. He observed three cases of acute ascending paralysis following longstanding chronic cystitis. Urinary paraplegia, as he calls it, he states has been recognized since 1833, when Stanley made a communication to the Medico-Chirurgical Society; but Stanley's case seems to have been different. In Walker's[13] case, the patient had a stricture of the urethra and chronic cystitis, and during an exacerbation he developed during a night numbness in the feet and some loss of power in the legs. The following morning he had paresis of the limbs up to the thighs. By the evening of the same day the paralysis had extended upward to the arms and thorax, and breathing was embarrassed. The symptoms rapidly progressed, and death occurred eighteen hours after their onset. So far as the author could remember, the catheter or bougie was not used during the last illness.

---

[10] Babinski: Semaine méd., 1909, No. 1.
[11] Oppenheim: Lehrbuch.
[12] Frank, E.: Berl. klin. Wchnschr., June 21, 1909, p. 1154.

In Case 2 the patient had chronic cystitis. December 31, he had acute exacerbation of his bladder trouble with malaise and some fever, but the symptoms were not urgent. On the morning of January 4 he experienced uncomfortable sensations and feebleness in the lower limbs. By midday he had paraplegia. He died forty-eight hours after the first indication of paralysis.

In Case 3 there had also been chronic cystitis. The extension of the paralysis upward was rapid. Death occurred eighteen hours after the first appearance of paralytic symptoms.

Necropsy was not obtained in any of the three cases.

The literature referred to by Walker is not extensive. In addition to Stanley, who in 1833 directed attention to urinary paraplegia and explained it as reflex paralysis, Troja is said by Leyden to have stated that violent inflammation of the kidneys may produce an irritation of the nerves of the kidneys and the inflammation may extend to the spinal cord. Guel[14] upheld the nervous reflex view of urinary paraplegia. Leyden[15] believed that the lesion was myelitis. He assumed that the inflammation first passed to the nerves and neuritis so produced may lead to myelitis.

---

[13] Walker, T. J.: Tr. Clinical Society of London, xxxviii, 80; Lancet, London, March 11, 1905, p. 636.

[14] Guel: Guy's Hosp. Rep., 1861.

[15] Leyden: German Clinical Lectures, New Sydenham Society.

339

Fenwick accepted Leyden's views, but there seems to be little or no pathologic study of these cases.

A case of rapidly fatal ascending paralysis in a man with pyuria and prostatic hypertrophy, but without necropsy, was reported by G. B. Buchanan.[16] It was suggested that the passage of an instrument in a patient, who had recently been losing health with his urinary system obviously diseased, might have induced an increased absorption of poisons into the blood, leading to a profound intoxication of the cord without giving any precursory signs.

Orr and Rows in several papers, and later in association with Stephenson,[17] have demonstrated that inflammation of the central nervous system is easily induced by infection of the ascending lymph-stream of nerves, and that the spinal cord and brain are exposed to infection in this way. Their experimental work has shown that infection of the lymph-system of peripheral nerves causes an ascending neuritis which spreads upward to pass over the posterior root ganglia and along the spinal roots to the cord. Any part of the central nervous system may be attacked by organisms or toxins passing up the nerves from infective foci, and the ultimate result of infection may vary according to the quality of the exciting cause.

The case which I report in this paper should, I think, be explained in the light of Orr and Rows' findings, and the myelitis should be regarded as secondary to the perirectal infection. It is not impossible that the liability to myelitis was increased by the fright which the man experienced. Ascending neuritis, however, has always been regarded as very uncommon.

2046 Chestnut Street.

---

[16] Buchanan, G. B.: Brit. Med. Jour., Jan. 20, 1906, p. 145.

[17] Orr, Rows and Stephenson: Review of Neurology and Psychiatry, July, 1913.

Reprinted from The Journal of the American Medical Association
Oct. 31, 1914, Vol. LXIII, pp. 1546-1549

Copyright, 1914
American Medical Association, 535 N. Dearborn St., Chicago

[Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 41, No. 9, September, 1914.]

SYMMETRICAL PAIN IN THE HEEL FROM EXOSTOSIS OF THE OS CALCIS

By William G. Spiller, M.D.

Dr. Spiller said there were many causes of pain in the heel and while exostoses of the os calcis are recognized they probably occur with considerable frequency and this cause of pain may readily be overlooked. As illustrative of this condition he reported the following case.

C. A. F., male, 57 years of age, has had two or three distinct attacks of gout in his lifetime treated with great benefit by Dr. C. A. Fife and Dr. David Edsall. His last attack occurred two years ago. He has had chronic disease of the right knee joint for many years. He is a stock broker and has stood much until pain in the feet compelled him to sit more. In January, 1914, he began to complain of pain in his heels, he may have had some pain before this time, but the pain in the right heel was attributed by the patient to the chronic affection of the right knee, and the pain in the left heel was supposed to be from extra weight placed upon this heel in attempt to relieve the right lower limb. He recalled that he had had discomfort in his feet which he attributed to his shoes and had tried to make these more comfortable. During the early winter he had done a little more walking than previously. All venereal disease was denied. It was thought that the pain might be from flat foot and he wore casts in his shoes but was made worse by them. The pain sometimes went above the ankles. It was most severe in standing and walking and was very slight when he was sitting, although he has had some pain in the heels when in bed. He consulted Dr. Spiller, April 1, 1914. An area of much tenderness to pressure was found on the sole of each foot, symmetrically situated at the inner side of the heel. No tenderness was obtained by lateral squeezing of either foot. Tactile and pain sensations were normal in the feet. The Achilles reflexes were normal. Dr. Spiller requested the patient to have X-ray photographs taken of the feet, and this was done by Dr. Pancoast. A spicule of bone was revealed extending downward from each os calcis.

343

[Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 40, No. 10, October, 1913.]

CEREBRAL PALSIES WITHOUT DEMONSTRABLE ANATOMICAL FINDINGS[1]

By JOHN H. W. RHEIN, M.D.

PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM AT THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE, NEUROLOGIST TO THE HOWARD HOSPITAL, PHYSICIAN TO THE PHILADELPHIA HOME FOR INCURABLES, PHILADELPHIA, ETC.

(*From the Home for Incurables, and the Laboratory of Neuropathology at the University of Pennsylvania.*)

Cases of cerebral palsies without demonstrable lesion in the brain and without implication of the pyramidal tracts have been frequently reported. The cases however have by no means a common pathology. Perhaps the most usual form of these palsies consists of hemiplegia of toxic origin, such as those that occur after or during nephritis (Jacobson (1), Brodier (2) and others); or during pulmonary tuberculosis, pneumonia or as the result of lead poisoning and puerperal infection (Jacobson), or in alcoholics (Eichorst (3)). They may be also the result of disturbances of the circulation consisting of changes in blood vessels (Hochhaus (4) and Wernicke (5)); or due to vasomotor disturbance of toxic origin causing fugacious edemas (Le Calvé (6)).

While the aim of this paper is to call attention especially to the cases of infantile cerebral palsies without a clear-cut demonstrable lesion it is impossible to exclude from brief reference, for the purpose of elucidation, these cases, as well as those in which a lesion of the brain is found but in which at the same time no degeneration of the pyramidal tracts can be demonstrated. Such for example are the cases reported by Binswanger (7), Babinski (8) and Oppenheim (9), in which tumors of the brain have caused paralysis but in which degeneration of the pyramidal tracts has been lacking.

In addition to these there is also a group of cases of hemiplegia without disease of the pyramidal tracts in which is found in the basil ganglia a lesion consisting of lacunae described by Marie (10) under the name of état criblé, instances of which have been reported by Mikulski (11). In this group may be included the cases exhibiting lesions of the temporal lobe (Mikulski), of the lenticular nucleus (Wilson (12) and Oppenheim (13)), of the caudate nucleus and optic thalamus (Hoestermann (14)), of the optic thalamus (Bischoff (15), Nothnagel (16), Wernicke (17) and Oppenheim (18)) and the case cited by Marie and Guillaine (19) of lesion of the red nucleus with secondary degeneration of the tegmentum, the cerebellar peduncle, and posterior longitudinal bundle.

---

[1] Read at the thirty-ninth annual meeting of the American Neurological Association, May 5, 6 and 7, 1913.

There is then beside these a large group of cases that are described as infantile cerebral palsies in which the symptoms are characteristic and comparatively uniform in which again however the pathology is varied.

Excluding the cases of porencephalon and atrophy in the motor and other regions, associated with degeneration of the pyramidal tracts there is a comparatively small number of cases on record in which there is little or no demonstrable lesion either in the cortex or the motor tracts of the spinal cord.

In a number of these cases no true degeneration is found in the pyramidal tracts but a fineness of the fibers (Spiller (20), Binswanger (21)); or a scarcity of fiber (Ganghofer (22)), or incomplete myelination of the fibers (Haushalter & Collins (23)), or agenesis or imperfect development of the pyramidal tracts (Spiller (24), Ganghofer, Steinlechner-Gretschischnikoff (25) and Otto (26)), or finally atrophy of the pyramidal tracts (Ganghofer, Steinlechner-Gretschischnikoff).

Cellular changes in the cortex more or less marked have been described in some cases, the exact significance of which I believe is still open to discussion.

In Otto's cases there was a lack of development of the cortical cells especially the large pyramidal cells, and an entire absence of Betz cells. The absence of secondary degeneration in the pyramidal tracts is difficult to explain in this case if we accept as true that the fibers of the pyramidal tracts are directly connected with the Betz cells.

In one of Rolly's (27) cases there was some ganglion cell degeneration of moderate grade, added to proliferation of the glia and small vessels. Otherwise except for a slight external hydrocephalus there was nothing abnormal.

In some of the cases the cells of the cortex show little or no change. In a second case cited by Rolly of bilateral spasticity in a child who died at the age of 2 months there was nothing pathological except a proliferation of the glia and blood vessels.

In Haushalter and Collin's case there was no abnormality of the Rolandic cortex and the pyramidal tracts were normal except for incomplete myelination.

In Ganghofer's case the findings were negative except in one there was a scarcity of fibers observed.

In the three cases described by Spiller very slight pathological changes were found. In one of his cases an idiot with contracture of the feet, and inability to support the body or to use the hands, the brain showed no gross lesions. The Betz cells were not numerous and were smaller than normal and the fibers of the pyramidal tracts were remarkably fine.

In a second case, an idiot who could not talk, walk, or support his body and who was probably spastic and had increased reflexes in the legs but not in the arms which could be used, there was no gross or microscopic change in the brain or cord.

The third case was that of a girl who could not walk or talk, nor could she use the hands in voluntary effort. The Betz cells were numerous and showed change which may have been due to post mortem degeneration. The cells of the spinal cord were normal. The fibers of the crossed pyramidal tracts of the cervical region were very fine and finer than the columns of Goll or of the pyramidal tracts in a normal child of the same age.

This case he looked upon as one of agenesia of the pyramidal tracts as was true he believed of his first case. In his second case he inclined to the belief that the nerve fibers in the pyramidal tracts "had not grown downward into the cord in normal numbers so far as the lumbar region, although the cervical region was well supplied."

Railton (28) described a case of spastic hemiplegia in an idiot of 3 years in which there was a depression forming a shallow groove parallel with and involving the fissure of Rolando. Microscopically the pons, medulla and cord were perfectly normal.

There was some diminution in the number of the large ganglia cells and increase in the neuroglia only.

More recently changes in the cells have been noted by Finkelnberg (29), Spielmeyer (30) and Hoestermann which they believe explain the cause of the paralysis as well as the preservation of the pyramidal tracts in these cases.

Finkelnberg's case was one of infantile spastic paraplegia which beside a moderate degree of hydrocephalus showed the presence of an atrophy of the cortex in the motor region with preservation of the large ganglion cells (Riesenpyramidenzellen) and in which secondary degeneration of the pyramidal tracts was absent. The cells in the cortex in the first to the fourth layers were diminished in number and irregularly arranged, while the Betz cells were normal. He did not believe that the hydrocephalus explained the degeneration of the cortex but believed that the atrophy of the cortical layer produced a functional disturbance affecting the motor conductibility, at the same time leaving the trophic influence intact.

Hoestermann cited a case of spastic diplegia in a woman of 34 years who had been an idiot and an epileptic since birth in which there was porencephalon of the right occipital lobe and some atrophy of the entire right hemisphere. There was a sclerotic atrophy of a great part of the cortex in the temporal, parietal, and occipital lobes. The spinal cord was practically intact.

In the cortex there was an irregular placing of the small pyramidal cells extending from the layer of the large ganglion cells to the molecular layer, consisting of a slight atrophy of the cells generally but which was very intense in the molecular layer. The large ganglion cells showed some irregularity but were in a normal position on the right side though not on the left. The pyramids in the medulla oblongata were normal except the left which was slightly lighter in color than the right. The von Monakow's bundle was not diseased.

In a second case of idiocy with convulsions, left-sided spastic palsy and inability to walk or talk, there was found microgyria. The small pyramidal cells in the right central convolution were reduced in number and were irregularly placed. There was no diminution in the large ganglion cells and these were normal. The left central convolutions were normal.

The left pyramidal tract was smaller than the right and smaller in proportion than the posterior columns, while the left anterior horns of the spinal cord were smaller than the right and poor in ganglion cells.

In a third case of Hoestermann, a young man, with strabisinus, left hemiplegia, spasticity and increased reflexes, the left hemisphere was slightly larger than normal and there was a porencephalic defect in the right temporal, occipital and frontal regions. The cortex in all its layers was poor in cells, particularly the molecular layers, while the small pyramidal cells were increased in number. The ganglion cells were present but were less numerous than normal. The spinal cord was intact.

In a fourth case of paralysis of all members in a girl of 9 years gradually developing since 4 years of age there was external hydrocephalus, hemorrhage in the right caudate nucleus and in the right optic thalamus of old date. There were, besides, irregular orientation of the pyramidal cells in the central convolution and almost complete disappearance of the fibers of the cortex as shown by the Weigert method.

The cord was negative except for an embryonal type of the anterior pyramidal tract in the thoracic region and a paleness of Helweg's triangular field in the cervical region.

Hoestermann believed that the atrophy of the cells in the upper layers of the cortex caused palsy while the preservation of the lower layers guaranteed intactness of the trophic influence upon the pyramidal tracts.

Spielmeyer described a case of hemiplegia dating 2 years before death. The right hemisphere weighed 200 grams less than the left. The red nucleus, the optic thalamus on the right side and the left side of the cerebellum were reduced in volume and showed evidences of secondary atrophy. The pyramidal tracts in the spinal cord were intact.

In the cortex on the right side the cells of the cellular layer to the depth of the third zone were completely absent but in the deep layers a few cells were seen. There were also some large and middle sized cells in the third layer but the large ganglion cells showed no difference on the two sides.

In a former paper (31) I reported a case of spastic paraplegia (Case 1) dating from childhood, in a patient who died at the age of 72 years. At the autopsy nothing was found abnormal macroscopically. A microscopic study of the cortex, pons, medulla oblongata, and spinal cord showed nothing pathological. The cells of the cortex appeared to be normal although no Betz cells were found in the sections from the paracentral region. In the spinal cord no degeneration was present and the examination was negative except for a fineness of the fibers of the crossed pyramidal tracts.

I shall in this report add two other cases, one of which was briefly presented to the Philadelphia Neurological Society on March 25, 1910 (35) and one for the privilege of reporting which I am indebted to Drs. Charles Frazier and William G. Spiller.

Case 2.—L. S. was admitted to the Philadelphia Home for Incurables May 12, 1895, and died October 26, 1909, at the age of 27. The family history was lacking, except that a brother and sister were healthy.

At the time of her admission she stated that she had been suffering from jerky movements as long as she could remember.

Examination made at the time of her admission resulted as follows: There were constant involuntary movements, choreiform in character in arms and legs and face and more marked on the left side. Any attempt at voluntary effort increased the movements. The left hand was in constant motion. The legs were rigid, especially the left, which could not be extended beyond a right angle. There was distinct weakness of both the left arm and leg. The grip in the right hand was much stronger than in the left. There was no palsy of the face, and the tongue protruded straight and in the median line.

The knee jerk was increased on the right side but could not be elicited on the left side on account of the rigidity. There was an attempt at ankle clonus on the right side.

The pupils were equal and reacted to light. The speech was a little hesitating. She walked with distinct weakness of the left leg, the weight of the body coming down on the left toe.

The patient was undersized and while the head was small it was not out of proportion to the rest of the body. The bitemporal diameter was 11.5 cm., the biparietal 13 cm., the occipito-frontal 18 cm., occipito-mental 20.5 cm., trachelo-bregmatic 16.5 cm.; and the occipito-frontalis circumference 50 cm.

The autopsy revealed nothing macroscopically amiss in the brain or spinal cord. The weight of the brain after having been in formaline for about two weeks was 1,167 grams. The average weight of the normal brain being 1,230 to 1,350 grams, taking into consideration the fact that the brain had been in formaline for 2 weeks, it could be estimated that the brain weighed considerably less than the average. The convolution of the brain showed no deviation from the normal.

Sections were made from the paracentral regions, from the pons medulla and from the cervical, thoracic, and lumbar regions of the cord.

The pia in the paracentral regions was thickened, and infiltrated with large mono-nuclear round cells, and connective tissue cells, the latter predominating. There was some peri-vascular distention and slight peri-vascular round cell infiltration in the superficial layers of the cortex in this region. The Betz cells were numerous and stained well, and the other cells of the cortex could not be said to be diminished in number or altered in their orientation.

No pathological change could be found in the white matter of the pons, medulla oblongata or spinal cord, either by the Weigert method or with the nuclear stain. The pyramidal bundles in the pons were perhaps a little smaller on the right side than on the left but otherwise no abnormality was noted.

The cells in the anterior horns of the spinal cord stained well and showed nothing abnormal except that in many there was a considerable amount of yellow pigment.

In a word there was no demonstrable pathological condition to account for the spastic diplegia dating probably from birth or at least early infancy.

Case 3. B. J. About 10 years of age, died 2 days after a subcutaneous incision of the plantar fascia and Achilles tendon. The pathological diagnosis after autopsy was chronic mitral and bicuspid endocarditis, broncho-pneumonia, fatty degeneration of the kidney and liver. The patient was operated on at the University Hospital by Dr. Charles Frazier, having been transferred to this institution temporarily from the Vineland Training School.

The patient was a large plump boy with low mentality since birth. The facial expression was vacant, and he did not talk. Both arms were spastic, but he used the arms freely, grasping with each hand fairly well. It was impossible however to fully extend the elbow joint on either side. At rest the fingers of the right hand were extended. The bicipital jerks could not be elicited.

The lower limbs were somewhat smaller than normal from the knees down, and he was unable to stand. There was talipes-equinovarus on both sides, and also a marked contracture of the Achilles tendons. The ham-string tendons were slightly contractured. The patient was able to flex the hips well but could not extend them fully.

The brain was quite small, measuring in its long diameter but 6 inches. Twenty-seven brains at the neuropathological laboratory of the University of Pennsylvania were measured and it was found that the average length was a little over seven inches. No abnormality of the convolutions was observed and a cross section of the brain was apparently normal in appearance.

Sections were made from the right and left paracentral regions, the basal ganglia on one side, the optic nerves, the medulla oblongata, the cervical, thoracic and lumbar regions of the spinal cord and of the anterior roots from the cervical and lumbar regions, and these were stained by the Weigert method and by thionin and hemalum.

The paracentral regions. By the Weigert method no degeneration of the white matter could be demonstrated. There could be seen however in the superficial layer of the cortex a fine network of fibers and nuclei.

By the nuclear stain the pia was found to be thickened in places and especially over the sulci, where it was markedly so. The process consisted of an increase of connective tissue fibers which were studded with numerous connective tissue cells, some mono-nuclear round cells and some red blood corpuscles. In other places the pia was slightly thickened but was not the seat of any disease process. In the cortex of the paracentral region there was considerable peri-vascular dilatation but no infiltration of round cells in the peri-vascular spaces was found.

By the thionin stain, many of the Betz cells in the left paracentral region stained normally. Some however stained poorly and showed other signs of degeneration. Some of the cells were swollen, had lost their processes and the nucleus had disappeared. The Betz cells seemed to be present in normal quantities. Sections from the right paracentral region showed practically the same process except here and there degenerated Betz cells were more numerous.

The cells in the other layers of the cortex appeared to present no abnormality as to position and numbers on the right side.

The optic chiasm and the optic nerves took the stain normally. The basal ganglia showed microscopically nothing pathological.

Sections from the medulla oblongata and from the cervical and lumbar regions of the cord were entirely negative with the exception of some insignificant changes of the cells of the anterior horns in the thoracic region consisting of atrophy of the pigment and displacement of the nucleus.

There was some slight degeneration of the anterior roots in the lumbar region. The pia of the cord was not diseased and no cause could be found for the degeneration of the anterior roots.

The pyramidal tracts of the cord stained well and showed no evidence of degeneration.

In this case the degeneration of the Betz cells was hardly extensive enough to explain a condition which had existed since birth, and the process described was not one which suggested that it had existed for a long time.

No satisfactory explanation of the paralysis has been offered in the cases in which the pyramidal tract is apparently normal and in which insignificant or no lesion is found in the brain.

The part played by the pyramidal tract in these cases presents an interesting problem. Intact pyramidal tracts imply that they are capable of transmitting motor impulses and in those cases in which the pyramidal tracts are normal, a cause for the palsy must be searched for above the spinal cord or even above the medulla oblongata and the pons.

It is reasonable to suppose that the cells of the cortex may be at fault, and disease of the cortical cells may occur without causing degeneration of the pyramidal tracts. That this is true has been shown by Binswanger who cited a case of tumor situated in the central convolution, one of tumor of the dura, and one in which there was a focus of softening in the cortex, with intact pyramidal tracts.

Babinski also cited a case of tumor of the motor cortex with intact pyramidal tracts. He believed that the irritation of the pyramidal tracts was sufficient to cause paralysis, but not intense enough to give rise to secondary degeneration.

Finkelnberg advanced the theory that the cause of the paralysis in his case was to be found in the atrophy of certain cortical cells which presided over function, while the preservation of the large ganglion cells (Riesenpyramidenzellen) insured a trophic influence upon the pyramidal tracts.

A similar explanation may be applied to the case described by Spielmeyer under the name of intracortical hemiplegia, above cited.

Hoestermann's cases may be cited as supporting this theory also. He emphasizes the importance of a careful histological study of the cortex, especially in the central convolution. He believed that when by atrophy the large ganglion cells (Riesenpyramidenzellen) are isolated from other cortical cells they are unable to exercise a functional influence upon the pyramidal tracts but the intactness of the large ganglion cells guarantees a trophic influence upon these tracts.

Another explanation he finds in the fact that different parts of the brain develop independently and these later become associated with each other, and hand in hand the function becomes established. If a lesion occurs before the functional differentiation but after the conclusion of the anatomical differentiation the function is destroyed, and finally if the organ is completely developed and both functional and anatomical differentiation is completed there occurs both a functional and anatomical defect.

The role of the optic thalamus must be considered at least in some of the cases of infantile cerebral palsies.

Bischoff (32) believed that the symptoms of infantile cerebral palsy can be caused by disease of the optic thalamus, and that an implication of the pyramidal tracts is not essential. Bischoff has shown that destruction of motor functions may be caused by cutting off of the sensory stimulation from the optic thalamus to the motor cortex. The optic thalamus appears he claims to be associated functionally with certain zones of the cortex by means of projection fibers.

Recently S. A. K. Wilson has described cases of progressive symmetrical lenticular degeneration, presenting bilateral involuntary tremor of all the limbs, rythmic usually, associated with spasticity of the limbs and face, dysarthria, dysphasia but little or no true paralysis. There is neurogliar proliferation, the glia replacing cells and fibers. There is also cavity formation and atrophy of the caudate nucleus. It is an extrapyramidal motor disease. Cirrhosis of the liver is a constant condition.

As the basal ganglia in my second case were normal it cannot be compared to this group of cases. Nor is there any resemblance to the cases of Oppenheim (33), which he described as pseudobulbar palsy occurring in children and exhibiting double athetosis, rythmic tremor, associated movements without sensory disturbance or implication of the intelligence. Vogt (34) found in these cases a marble-like appearance together with atrophy of the caudate and lenticular nuclei.

In my own cases with the exception of the first case in which no Betz cells were found, no change in the cells of the cortex could be demonstrated. The explanations offered therefore by Finkelnberg, Hoestermann and Spielmeyer do not satisfactorily apply to these cases.

What the cause of the paralysis was in my cases is yet undetermined. Whether it be due to some deficiency of nerve cell which baffles our present methods of study to demonstrate; whether it be due to faulty connection between various layers of the cortical cells; whether the nerve fibers in the pyramidal tracts have not developed in sufficient numbers in the lower levels of the cord (Spiller); or whether the fineness of the fibers interferes with their perfect function by offering undue resistance to the transmission of the motor impulse from the cortex and thus preventing the full motor discharge to reach the periphery (Rhein), is still a matter for study in the future to determine. It is difficult to avoid the conviction that one must look, for the explanation of these cases, to some quality of nerve cell or fiber which cannot by our present methods be demonstrated.

1. Jacobson. Deutsch. Ztschr. f. Nervenh., 1893, Vol. IV, p. 235.
2. Brodier. Arch. gen. de méd., 1900, p. 1008.
3. Eichhorst. Virchow's Arch. f. path. Anat., 1892, Vol. CXXIX, p. 140.
4. Hochhaus. Deutsch. med. Wchnschr., 1908, Vol. II, p. 1657.
5. Wernicke. Gehirnkrankheiten, Vol. II.
6. Le Calve. Arch. gen. méd., 1903, Vol. X, p. 577.
7. Binswanger. In Hoestermann.
8. Babinski. Bull. et mém. Soc. méd. d. hôp. de Paris, 1899, p. 342, and Revue de neurol., 1906, Vol. XIV, p. 693.
9. Oppenheim. In Hochhaus.
10. Marie. Rev. de méd., 1901, Vol. XXI.
11. Mikulski. Arch. f. Psychiat., 1909–10, Vol. XLVI, p. 434.
12. Wilson. Brain, 1912, Vol. XXIV, p. 295.
13. Oppenheim. In Bischoff.
14. Hoestermann. Arch. f. Psych., 1912, p. 40.
15. Bischoff. Arch. f. Psychiat., Vol. XVII, p. 221.
16. Nothnagel. In Bischoff.
17. Wernicke. In Bischoff.
18. Oppenheim. In Bischoff.
19. Marie and Guillaine. Nouv. iconog. de la Salpétrière, 1903, p. 80.
20. Spiller. University of Pennsylvania Med. Bull., 1905.
21. Binswanger. Virchow's Arch., 1882, p. 427.
22. Ganghofer. Ztschr. f. Heilk., Prague, 1896, p. 303.
23. Haushalter and Collins. Compt, rend. Soc. de biol., 1905, p. 223.
24. Spiller. Jour. Nerv. and Ment. Dis., 1898, p. 81.
25. Steinlechner-Gretschischnikoff. Arch. f. Psychiat., 1886, Vol. XVII, p. 649.

26. Otto. Arch. f. Psychiat., 1891–2, Vol. XXIII.
27. Rolly. Deutsch. Ztschr. f. Nervenh., 1901, p. 152.
28. Railton. Brit. med. Jour., 1892, Vol. I, p. 441.
29. Finkelnberg. Deutsch. Ztschr. f. Nervenh., 1913, p. 163.
30. Spielmeyer. Munchen. med. Wchnschr., 1906, Vol. II, p. 1404.
31. Rhein. Am. Jour. Med. Sc., 1909, Dec.
32. Bischoff. Jahresb. f. Psychiat., 1897, Vol. XV.
33. Oppenheim-Spiller. "Progressive Medicine," 1912, p. 307.
34. Freund and Vogt. Jour. f. Psychiat. u. Neurol., 1911, Vol. XVIII.
35. Rhein. JOUR. NERV. AND MENT. DIS., 1911.

Reprinted from the New York Medical Journal for
May 2, 1914.

THE DIFFERENT THEORIES OF APHASIA.*

By Charles K. Mills, M. D.,
Philadelphia,
Professor of Neurology, University of Pennsylvania.

That interest in the subject of aphasia has suffered no abatement, notwithstanding its frequent discussion since Marie's announcement of his view of the importance of mental deficit in the explanation of aphasic phenomena, is evidenced by the fact that at the recent International Medical Congress, held in London in August of last year, some of the greatest men in the field of neurology gave their attention and time to the consideration of the cerebral disturbances of speech. The members of this society, especially those connected with the Philadelphia General Hospital, with its wards rich in organic nervous disease, are especially fitted by their opportunities and investigations to contribute to the solution of disputed questions concerning aphasia. I regard it therefore as fortunate that the council of the society has deemed it worth while to suggest a symposium on affections of speech of cerebral origin, although these have not infrequently claimed attention in recent and more remote periods of our society's history. Some scientific basis must exist for the views of experienced and enlightened observers, and for this reason I have no doubt that the apparently conflicting data and theories of neurologists of such distinction as Déjerine, Marie, von Monakow, Liepmann, Moutier, and Dercum, must in large part be reconcilable. Divergence of opinion on this subject as on so many others is largely due to method of interpretation. In opening

---

*Remarks made in opening a discussion on aphasia at the meeting of the Philadelphia Neurological Society, February 27, 1914.

Copyright, 1914, by A. R. Elliott Publishing Company.

this discussion, therefore, I shall not so much attempt to give new facts and ideas as endeavor to show the possibility of harmonizing some apparently widely separated opinions.

The three theories regarding aphasia requiring attention in this discussion are: 1, The so called classical view based on the discoveries of Broca, Wernicke, and others; 2, the mental deficit theory of Marie; and, 3, Liepmann's doctrine of apraxia as applied to aphasia. The diaschisis of von Monakow must also receive consideration.

The older or so called classical view of aphasia is one that recognizes a subdivision of the cerebral zone of speech into at least two subareas, one sensory and the other motor. According to some authorities, a third area, which might be called conceptual, although the term is open to objection, is intercalated between the sensory and motor speech regions. Accepting the subdivision into sensory and motor areas, the sensory region has generally been recognized as subdivided into at least two parts, one auditory and the other visual, these being respectively places of concentration or storage of auditory and visual images.

Much objection has been made at times to the use of such expressions as centres and memorial images, because of their supposed lack of definiteness; but it matters little how these centres of function are designated or explained, if the fact is recognized that they are in some sense foci of activity resulting in special phenomena.

Mistakes are sometimes made by the opponents of localization, and perhaps also by the localizationists themselves, in giving a loose interpretation to the conception of centres. Every centre is simply one of the foci of a mechanism which has many associations and interrelations within itself and with other parts of the brain. Nevertheless, it has its particular purpose in the general machinery of which it is a part—a purpose which can be determined by investigation.

In *Science* for April, 1912, F. H. Pike has put this matter of localization of function so neatly and clearly that it seems worth while to make one or two citations from him in this connection:

> It is true that Marie and von Monakow have shown that certain of the more or less current conceptions of focal or insular representation of cerebral function, particularly those concerning the speech centre, are no longer tenable, but neither Marie nor von Monakow has denied that certain definite fibres arise from definite circumscribed areas of cells in the cerebral cortex and run to certain definite end stations. Indeed, no point of nervous anatomy or physiology seems better established than this. And stimulation of a definite, circumscribed area of the cerebral motor cortex of any one animal always elicits a response of a definite group of muscles, and never of any other groups. This definite, circumscribed cell area constitutes the focal or insular motor representation in the cerebral motor cortex of this particular group of muscles. Such a circumscribed area, frequently marked off from surrounding cells by a boundary of nonnervous tissue, is commonly known as a motor centre.
>
> It would, however, be an error to suppose that this group of cells is an isolated group. It has, through afferent association neurones, connections with practically every portion of the cerebral hemisphere of the same side; through commissural neurones, with practically every portion of the opposite side, and through afferent [efferent?] projection neurones, it is brought into relation, directly or indirectly, with the cerebellum, spinal cord, and other structures. The circumscribed area of cells thus becomes a part of an extremely complex and extensive *motor system,* but in such participation it loses nothing in definiteness of location or of function. The conception of a circumscribed motor centre must be superseded by the conception of a motor system or mechanism. It is to such a mechanism that we may apply the conception of integration as developed by Sherrington. Integration is essentially a dynamic rather than a morphological process; but integration implies a certain definiteness of relationships, morphological as well as dynamic, throughout any one process. We have already stated that stimulation of the particular group of cells always evokes a response of a particular group of muscles—contraction of one set and relaxation of their antagonists. And since this phenomenon of group movement is constant from day to day or year to year in any one individual, we are justified in assuming that a certain rather constant morphological mechanism integrates certain rather constant relations in time and space to a fairly constant result.

This is the essence of the modern dynamic view of localization of function.

The principles which are illustrated by this discussion of motor centres and integrating mechanisms, are equally applicable to the explanation of the phenomena of sensation and even those of mentation. Pike says again:

> For my part I find it impossible to gain a clear idea of how the brain functions as a whole in motor processes. I do believe, however, that we may gain a certain degree of clearness of ideas if we suppose that certain definite circumscribed cell areas, and no others, acting through their association tracts, may evoke a definite motor reaction, and no other motor reaction. Similarly, I find it extremely difficult to see how the brain, acting as a whole and without reference to circumscribed cerebral areas or to integration systems involving two or more such areas, may give rise to a mental process. I can picture to myself a conceivable way in which several cell groups or systems, acting together in a particular manner and without special reference to the rest of the brain, may give rise to a particular mental process or conception made up of certain definite mental elements which are related in a definite way. Other parts of the brain may subsequently be involved in succeeding mental processes, but a definite order of succession may well be followed.

As regards the motor region for speech, the view maintained since the time of Broca is that a certain cortical area, usually defined as the hinder part of Broca's convolution or of this and the preinsula, is in some especial way concerned with emissive speech. Many explanations of the manner in which this so called motor centre for speech performs its important functions have been advocated. By some it has been called a zone for motor images; by others an arranging centre or place where language is prepared for emission by the motor projection system. Broadbent, with something of this kind in view, called it a "propositionizing centre"; but whatever the definition suggested or the position taken, until the time of Marie all writers and investigators held to the view that Broca's centre was the seat of some special activity concerned with speech.

Briefly stated, the views of Marie are in the first place, that auditory, visual, and motor speech centres in the sense usually accepted do not exist; that aphasic phenomena are not due to interference with auditory, visual, or other images, but to intellectual deficit, this deficit causing difficulty in the comprehension of speech and interfering with numerous didactic processes. Marie also holds that Broca's convolution takes no part in the function of speech, nor its lesions in the production of aphasia. This is perhaps the most startling of his assertions. He admits that sensory aphasia, motor aphasia, and anarthria are clinical facts, but explains these, especially as regards motor aphasia, differently from his predecessors—asserting that motor aphasia is nothing but sensory aphasia with the addition of anarthria. Anarthria, as he understands the term, is due to lesion of the lenticula and its environment. According to him the regions, lesions of which produce true aphasia, are all included in Wernicke's zone.

Mental deficit is present in all cases of true aphasia. We have mental deficit in which the defect in language comprehension of sensory aphasia predominates, mental deficit in which the inability to comprehend the meaning of words and the method of using language predominates, and mental deficit in which both incapacities are commingled as in some forms of sensorimotor aphasia. The deficit is due to the destruction or disruption of clearly recognizable special cortical centres and associating tracts and the connections of such associated centres with the sensory and motor projection system and other regions of the brain.

The apraxia theory of Liepmann has lately come into much prominence. Speaking generally, apraxia is the inability to apply knowledge purposively, this lack being particularly manifested in skilled movements of all sorts. The motor apraxic, for instance, may be unable to use his upper or lower extremity in work which requires skillful adaptation of the muscular power which he possesses. The movements concerned with phonation, enunciation, and articulation cannot be so made as to yield intelligible speech, although the patient may be able to use the musculature, out of tune as regards speech, for other purposes like chewing, swallowing, or breathing. Aphasia defined in terms of apraxia is especially the loss of certain skilled acquisitions. A sensory and an ideational apraxia as well as a motor apraxia are recognized and many of the phenomena of aphasia are to be referred to their presence.

More clearly perhaps than any other theory suggested by writers on aphasia, that of diaschisis throws light upon disputed problems and brings more nearly together the views of different authorities as just presented. According to this doctrine enunciated by von Monakow, circumscribed lesions give rise to symptoms—speech disturbance, paralytic phenomena, etc.—much more marked than one would expect considering what is regarded as the special function of the part affected. Groups of associated neurones are injured and functionally affected because of destruction of some important part of the system. The disturbance of function is widespread as the connection of the parts affected are widely situated. Restoration in whole or part may take place as structures are released or new paths are formed.

One important fact emphasized by von Monakow and others is that lesions of the brain are rarely purely cortical. Cortex and subcortex are nearly always involved in a gross lesion. This being the case, it follows that in all lesions involving special centres like those which have been spoken of as centres for word seeing and word hearing, motor images, etc., not only is the circumscribed area of cortical gray matter which is supposed to be the particular rendezvous of the dynamism which gives the cortical focus its name, affected by the lesion, but all the parts anatomically and physiologically connected with it.

Accepting for the sake of argument the existence of a centre particularly concerned with the auditory comprehension of words and phrases, if such a centre is destroyed, even in the most circumscribed fashion, results of this destruction must extend to every part with which this centre is associated. The doctrine of diaschisis is based upon the recognition of integrating mechanisms. The theories to which I have called attention, it will be seen, can stand only on the basis of the admission of the existence in the human cerebral cortex of numerous centres. The whole fabric of focal diagnosis built up by the work of thousands of investigators in the last fifty years, would fall to pieces if we failed to recognize this fact.

The second fact which needs to be emphasized is that these so called centres, wherever located, have formed few or many associations with each other and with other regions, according to their character and importance. The idea of a centre of special function seems to be a bugbear to many who prefer rather to deal with vague generalizations about association phenomena. The true way to look at this subject is to accept both centres and associating mechanisms, and thus harmonize views which would otherwise get too far apart. The very existence of associations indicates something to be associated. Associating structures are not simply paths which come from nowhere and go nowhere. They are lines of connection between important structural and dynamic foci.

We can accept Marie's idea, that disturbance and the impairment in the comprehension of language are dependent upon intellectual deficit, but we must also accept the explanation of Déjerine that this deficit in its turn is dependent upon destruction or disruption of a mechanism which is made up of associated centres. To accept any other view would throw us back upon some ancient theological or metaphysical theory of the existence of mind outside of the body.

Those who oppose the idea of centres for auditory, visual, and other images, and invoke association to explain the facts of aphasia, in the act of doing this call to their aid the refinements of localization. At the same time that they would exclude from consideration centres for auditory and visual images, some of them discuss the contributory effects and the interrelation of centres for touch, taste, smell, and the like in the explanation of speech phenomena.

The phenomena of apraxia can be explained only by the acceptance of the idea of highly specialized centres linked structurally and functionally together. Liepmann recognized this and the further fact that these cortical dynamic areas reach over from one to the other in the performance of their functions. The entire idea on which the apraxia concept is based is one which must recognize an extraordinary development of cortical centres and elaborate neural associations which are the substrata of higher human acquisitions. It is these acquisitions which are interfered with in the apraxic, whether with regard to limb movements or speech movements or language comprehension, and every skilled acquisition is represented by a definite cerebral centre and integrating mechanism.

Too much emphasis may have been laid on the use of the word "motor" in the description of what is termed Broca's or motor aphasia. In a certain sense it is correct to retain this designation. Topographically, the so called motor centre for speech—by which I mean the hinder part of the left third frontal convolution and the anterior half of the insula—is in close proximity to the accepted motor region of the brain, and to structures like the lenticula and internal capsule, generally recognized as largely concerned with motor functions. While Broca's convolution and the preinsula do not constitute a sensory area in the same way as the centres and zones of the temporal and parietooccipital regions, this so called motor speech area is in a broad sense a region concerned with language comprehension, at least with the comprehension of the meaning of words and the method of using them, but in admitting this it must be understood that its function is entirely different from the functions of the centres in Wernicke's zone. Most writers on the subject of aphasia who believe in the classical theory, have more or less fully recognized the role just indicated as that played by Broca's area.

In connection herewith I might refer to one of the methods suggested for discriminating between motor and sensory aphasia, namely, that of Liepmann, although I am not sure that this suggestion can be fully endorsed. Liepmann says that while it may be admitted that some cases of motor aphasia reveal a "defect of intelligence," this defect is always of the nature of a failure to understand the meaning of words, whereas the failure of the typical cortical sensory aphasic is in understanding the sound of words. In the motor aphasic this "defect of intelligence," if present, indicates that he cannot associate quickly enough the words of a sentence with their corresponding inner meaning. (*Neurologisches Centralblatt,* p. 290, April, 1908.)

Motor aphasics may not understand the meaning of words, but to my mind their deficit is more than this—they do not understand the method of using words. It may also be questioned whether the sensory aphasic's deficit is simply a failure to understand the sound of words.

What then is the nature of the process which is interfered with, sometimes to the point of extinction, in cases of motor aphasia? It is most difficult to understand this matter and still more difficult to put the explanation in didactic form. This difficulty is the rock on which many of those who have attempted to explain the phenomena of aphasia have split.

In the first place the process which is interfered with is in the truest sense an intellectual one; in the second place it is as complex as language itself. Presuming a patient with Wernicke's zone intact and the cortical or corticosubcortical motor speech region destroyed, this patient, theoretically at least and to a large extent in fact, can understand what he hears or what he reads, this comprehension being disturbed only in an indirect way because of his deficiency by diaschisis. The essential thing is that he cannot speak, that he cannot translate what he hears and sees and thinks into vocal and articulative movements.

The motor aphasic, as all experience shows, apparently cannot recall all the words and phrases which he should use, but it can be demonstrated that this *Amnesia verbalis* is only a part of his deficit, and it may be doubtful whether it is a fundamental loss. For some reason, whatever his interior knowledge of names and the meanings of words and phrases may be, he cannot recall and use them for the purposes of speech.

At first sight the motor apraxia of Liepmann would seem to be a fairly good explanation of the aphasic's deficiency, as he has evidently lost the acquired skill which formerly enabled him to use for the purpose of speaking the musculature usually employed in speech. He is a motor apraxic, but this is not all. He is not only unable to use the motor executive apparatus for speech—which may be intact from his cerebral motor cortex to his bulbar nuclei and to the peripheral neuromuscular apparatus itself—but as some carefully studied cases show, especially those in which the motor aphasic has been retrained to speak, regaining in large part if not altogether the power to use his neuromuscular apparatus, he is still crippled, and it may be greatly crippled, in his powers of utterance. In several cases which I have carefully studied, it has become evident that the partially retrained motor aphasic is unable to understand the method of using language by putting together the elements of emissive speech in correct form, although he may have become competent to use separate words, or even to some extent phrases and sentences. For him the grammar of language has largely disappeared, it being understood that the word, grammar, is used in a very broad sense to apply to the former acquisitions in speech of the ignorant, the highly cultivated, and those of intermediate linguistic accomplishments.

For the motor aphasic the fabric of emissive speech in all its complexities has been shattered or destroyed. If he is an apraxic, his apraxia is more than simply motor. It is ideational and perhaps in part sensorial. In brief, he no longer comprehends the methods of expressing himself in speech, at least he cannot make use of these methods for the purposes of speech. He has become asyntactic.

Syntax in grammar refers to the process by which words are put in proper relation to each other in sentences so that the full meaning of the phrase or sentence is shown. This is exactly one of the things which a motor aphasic cannot do, and the defect is entirely different from the limitations of a sensorial aphasic and from the language disorder of an anarthric patient. His affection indicates an interference with an intellectual process of cortical or corticosubcortical origin if we keep in view the idea of integrating mechanisms. If a patient has a lesion producing this affection, it will interfere, not only with his powers of "propositionizing," as Broadbent would say, but also in less or greater degree with his readiness in comprehending or using language in any way. Remembering these facts, we can understand the interference with both language comprehension and language emission which motor aphasics show.

In the form of aphasia under consideration the difficulty is, as Déjerine has pointed out, that the patient does not know how to speak, that is, how to use his cortical and corticosubcortical motor executive apparatus, which may be unaffected by disease, although in some instances it may take part in such disease and thus give a more extensive symptomatology.

At one time it was the fashion to speak of a motor aphasic as an ataxic aphasic, but unless a broad and loose interpretation is given to the word ataxic, the explanation involved in this expression is not sufficient. It is not a question of disorder so much as of disablement, as Wyllie puts it, and therefore *Asynergia verbalis* as suggested by Ladame is more explanatory and intelligible. Certain things which formerly worked together to the end that the individual might speak, can no longer be properly harnessed together for the purposes of speech. The truth of the matter is, no single term is sufficiently expressive and descriptive to explain the disorder called motor aphasia; nor is any single theory sufficient for this purpose.

All who have seen many cases of aphasia, however, recognize the fact that in the clinical type called motor aphasia are included phenomena which cannot be explained on the doctrine that they are the result simply of sensory aphasia plus a paralytic anarthria, and I do not think we should accept any definition of anarthria which does not include the idea of paralysis. Uncomplicated true motor aphasia, as I have tried to show, gives symptoms radically different from those of sensory aphasia and sensory aphasia combined with paralytic anarthria. These symptoms are preeminently, according to all the light which we have with regard to cerebral phenomena, *cortical* in their nature. This is not to say that we may not have a subcortical motor aphasia, such as Déjerine has described under the name of pure motor aphasia. The phenomena of this form of aphasia are, however, the resultants of interference with structures associating cortical areas or cortical and other regions of the brain.

On the part of those who hold to the classical view of aphasia, the concession is necessary that the speech centre which I have just been discussing, and which simply for convenience is called the motor or Broca's centre, has a larger structural area than the hinder part of the left third frontal convolution. For a long time I have taught that the anterior portion of the insula must be included in this speech area, and I would go further than this. It is not impossible that it may also in some individuals take in a part of the first temporal convolution where this merges with the insula. The bearing of this concession upon the discussion in cases in which aphasia has been absent in lesions of Broca's convolution or present in the absence of such lesions is at once apparent.

In connection herewith the observations of E. A. Spitzka, showing the redundancy of the preinsula and its exposure because of this redundancy in the brains of some highly educated men, especially of those with an unusual gift of language, is of much interest.

Like Mingazzini and unlike Déjerine, I believe that the lenticula has some function connected with motor speech. It is a motor organ, and as Mingazzini also holds, functionally subdivided into separate centres concerned with movements of the face, the upper and lower extremity, etc. It is not improbable that its functions, in part at least, are concerned with emotional expression, whether this is exhibited in speech or in limb movements or in movements of any kind whatever. It is not in any way concerned with language comprehension. Its lesions render the patient's language less energetic and less fluent. Just as its lesions give rise to paralyses of the limbs which are partial and of a very special kind, so certain restricted lesions of the putamen of the lenticula, and especially of the left lenticula, may give rise to partial and peculiar affections of speech and expression.

S. A. Kinnier Wilson (*Brain*, XXXIV, 4, March, 1912) holds that the dysarthria present in his cases of bilateral lenticular degeneration was due to hypertonicity of the musculature of speech and is referable to the same cause as the dysphagia, tremor, weakness, spasticity or hypertonicity, contracture attitudes, contractures and emotionalism, the symptoms which on the positive side constitute his lenticular symptom complex. According to him the dysarthria and anarthria are not paralytic disturbances in the ordinary sense. With this view I agree, at least as I understand the matter. My own view is that destructive lesions of the lenticula cause what might be termed affective motor paralysis or paresis, although secondarily voluntary motor weakness results. There is an affective motor system as well as a voluntary motor system. The former acts on the latter both in an excitory and inhibitory way, that is, both as an energizer and regulator. As I have indicated elsewhere in my paper on the *Cerebral Mechanism of Emotional Expression,* there is a cortical affective motor zone or emotive motor zone. Speech movements, movements of swallowing, limb movements, and all forms of motor activity are influenced through this affective motor system which gives affective or emotional tone to voluntary movements. The cortical portion of this affective motor system is midfrontal, or midfrontal and prefrontal, and is connected by efferent or associative fibres not only with the thalamus but also with the lenticula. The latter Wilson does not appear to hold, if I understand his statements and his schema correctly.

The lenticula seems to me to occupy a position in the affective motor system comparable to that occupied by the thalamus in the great sensory pathway. This affective motor system communicates by the ansa lenticularis and other fibre systems with the cerebellorubrothalamocortical system and the cerebellorubrospinal system.

The steadying influence exerted by the affective motor cortex on the pyramidal motor system is interrupted in the lenticula when lesions occur in this ganglion, this interruption resulting in the disorders of speech and movement which give the lenticular syndrome of Wilson.

If the lenticula has nothing to do with the comprehension of language or the comprehension of the method of using language, but is a motor organ of particular type; and if it is true also that motor aphasia is a form of speech disturbance which includes a loss of language comprehension and yet one entirely different from that present in Wernicke's aphasia, it follows logically that motor aphasia cannot be the result of a lesion of Wernicke's zone plus one of the lenticula. It will be readily seen that the same reasoning would apply to a lesion of any part of the internal capsule purely executive motor in function.

The insula is part of Marie's lenticular zone, but if my contention be true that the insula or a part of it is linked with the hinder part of the third frontal convolution in the formation of the so called motor centre for speech, its lesions may give rise to the phenomena of motor aphasia. In my experience when the insula alone is affected, Broca's convolution escaping, the aphasic phenomena are not as persistent as when both are implicated in the disease. It is probable that the extent to which both the third frontal and the insula function as a language centre is conditioned by individual attainments in language.

In these remarks I have made no effort to add new facts to the data of aphasia. My object is simply to open the discussion by directing attention to some features of the subject most deserving of attention and most likely to attract polemical consideration. My views, however, are founded on personal observation as well as a study of the literature of the subject. The members of the society will recall my contributions to the subject of aphasia and those which I have made in collaboration with Doctor Spiller and others, most of which were presented originally either at the meeting of this society or of the American Neurological Association. I would especially direct attention to the articles and bibliography in my work on *Diseases of the Brain and Cranial Nerves,* etc., and to the article written in collaboration with Doctor Spiller on the *Symptomatology of Lesions of the Lenticular Zone with Some Discussion of the Pathology of Aphasia,* and to the article on the *Cerebral Mechanism of Emotional Expression* presented to the College of Physicians, March 6, 1912.

1909 CHESTNUT STREET.

[Reprinted from THE JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 41, No. 10, October, 1914.]

CONTUSION OF THE BRAIN[1]

BY JOHN H. W. RHEIN, M.D.

PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM AT THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE; NEUROLOGIST TO THE HOWARD HOSPITAL

Concussion is looked upon by many writers as a molecular vibration in the brain substance. On the other hand Kocher,[1] Keen[2] and others believed that there is an actual injury to the brain substance. In support of the former contention, the experimental work of Koch[3] and Filehne and that of Witkowski[4] on dogs shows that fatal concussion may be caused without more than evidence of hyperemia of the brain and it coverings.

Recently I have been called upon to examine a number of patients who had received injuries to the head and in whom symptoms of so-called concussion have lasted several days, also one patient with localizing symptoms who recovered, and one with localizing symptoms who died. I have been able to add to these a case kindly furnished me by Dr. W. G. Spiller. As a result of this experience I was led to make a study of the subject of contusion of the brain from a microscopic standpoint, as well as, in part, clinically. The number of cases is scarcely large enough to permit of any positive deductions, but some interesting features of the subject have been developed as a result of this study, the results of which may be looked upon as a preliminary report.

I found in the first place that the study of contusion of the brain is largely neglected by neurologists and on the other hand that comparatively little has been written about the subject even by surgeons. At first after reading Phelps's[5] paper on the subject in which he so ably and exhaustively deals with contusion and laceration of the brain I was tempted to drop the subject entirely. His paper however treats of the pathology of the subject from a macroscopic standpoint largely.

Little has been written since his paper published in 1893 and

[1] Read by title at the fortieth annual meeting of the American Neurological Association, May 7, 8 and 9, 1914. From the laboratory of Neuropathology, University of Pennsylvania, and the Philadelphia Polyclinic and College for graduates in Medicine.

this brief paper on the subject is warranted, I believe, by the fact that sufficient attention has not been given since to the subject by neurologists, whose assistance is or should be sought in each case before operation is decided upon.

Phelps analyzed 124 cases of brain injury occurring in his own experience. As a result of this study he stated that laceration and contusion were first in frequency and importance among all the injuries to the head. Contusion he stated may occur without lacerations and vice versa but they are usually associated. There were 28 cases of laceration without noticeable contusion in 58 cases, and but 10 cases of contusion without lacerations. By laceration he means a tearing of the tissue, the wound containing a coagulum beneath which is found a granular detritus of the brain structure. He described subcortical laceration as a simple extravasation of blood into the deeper tissues. In contusion he claims there is no solution of the continuity of the brain fibers but there are minute hemorrhages in the brain substance.

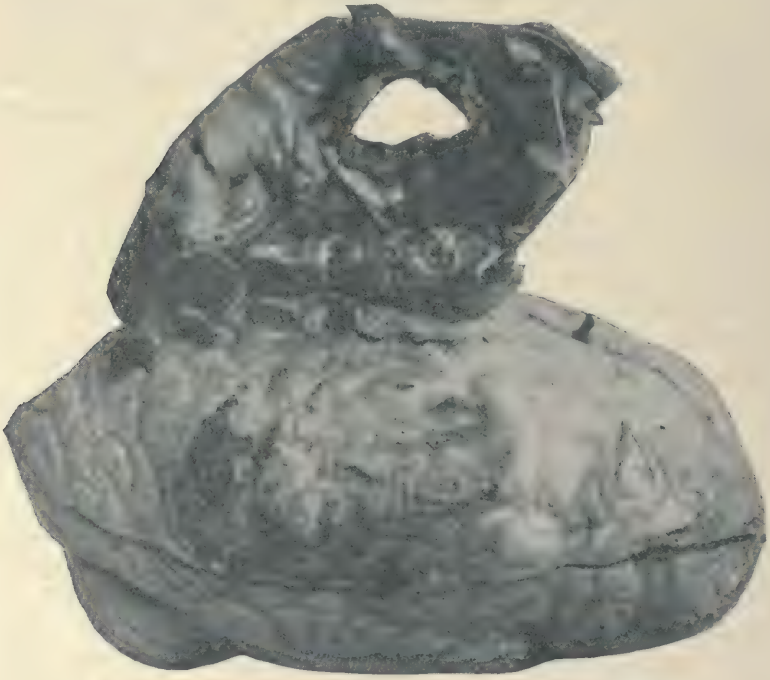


FIG. 1. Case 1. Showing subdural and subpial hemorrhage.

In Phelps's experiences the lacerations occurred more especially on the basal surface of the temporal and frontal convolutions but all parts of the brain may be affected, including the pons, cerebellum, the corpora striata and the gyrus fornicatus. Lesions may be superficial or deep, or may implicate the interior of the brain without reaching the surface. The membranes may or may not be involved and the pial hemorrhage may extend over large areas. The hemorrhage may be the only lesion in some places.

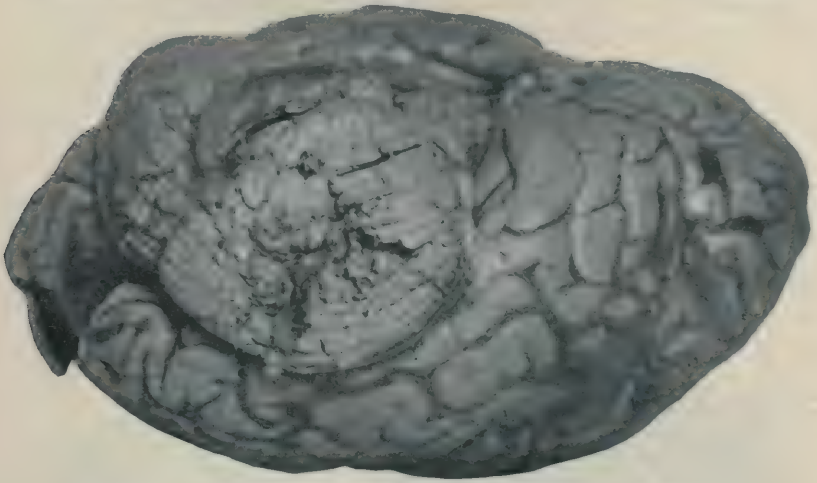


FIG. 2. Case 1. Showing area of softening in cerebellum.

Kocher (in 1901) has given the best description of the subject since Phelps's paper. He quotes six cases, five of them fracture of the bones of the skull and one, a case of marked contusion aggravated by excephalitis. Contusion of the temporal lobes, of the left frontal lobe and of the lower surface of the frontal lobe was observed in these cases caused by contre-coup and there was associated, epidural and sub-dural extravasation.

Page[6] in 1902 in discussing the subject of concussion of the brain in some of its surgical aspects described bruising of the brain tissue, not only at the seat of injury but also at a point opposite, due to contre-coup or the tearing of the walls of the brain from sudden displacement of the cerebro-spinal fluid from the lateral ventricle through the Sylvian aqueduct and fourth ventricle.

In the cases briefly to be described the chief points of interest are as follows: Extensive pial hemorrhage may exist without any injury to the cortex or cortical cells. Contused areas showed fairly sharply defined infiltration of blood with little demonstrable effect upon the adjoining cortical tissue. An inflammatory process was not found in the brain tissue. No evidence of a proliferative process was observed in that portion of the pia which was the seat of the hemorrhage. Injury to the brain appeared in some places to be due to hemorrhage extending from the pia. The cortical cells adjacent to the hemorrhagic infiltration of the cortex did not appear to show any evidence of degeneration.

The question arises, is it not advisable to drain the hemorrhagic area in the brain by an incision and thus relieve the pressure and ultimate destruction of the brain tissue which would ensue if the hemorrhage were allowed to remain? If the bruised areas were drained would not the tissues have less of a tendency

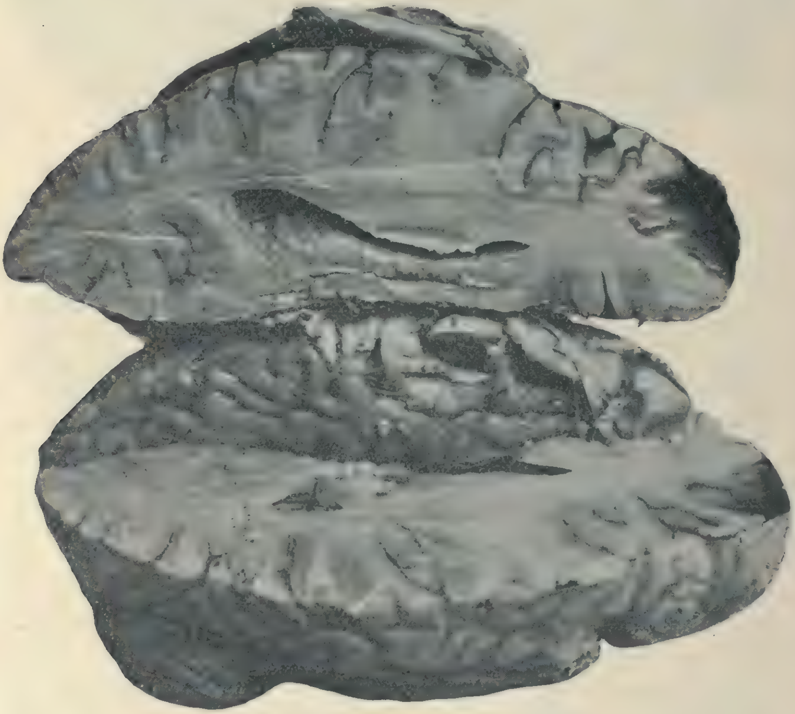


FIG. 3. Case I. Showing subpial hemorrhage which extruded into the superficial cortical layers.

to undergo softening and would not the relief of the pressure add to the chances of saving life?

CASE 1. E.McQ., aged 61, was admitted to the Philadelphia General Hospital, April 28, 1906, and died three days later. There is no history of injury but there is no doubt from the autopsy findings that she must have had some serious blow or injury before admission. The examination by Dr. Spiller was as follows: She had recurrent convulsive attacks involving the left side of the face in the upper as well as the lower distribution. The eyeballs were jerked to the left clonically and there was a slight jerking of the right eyelid in addition to the violent spasms of the left. The right arm was flexed at the elbow and the left arm and both legs were rigid at first. This was followed by clonic spasms in the left arm and both legs but not in the right arm. She was slightly stuporous between the convulsive attacks but replied to questions although unreliably. These convulsions occurred about every 10 or 15 minutes.

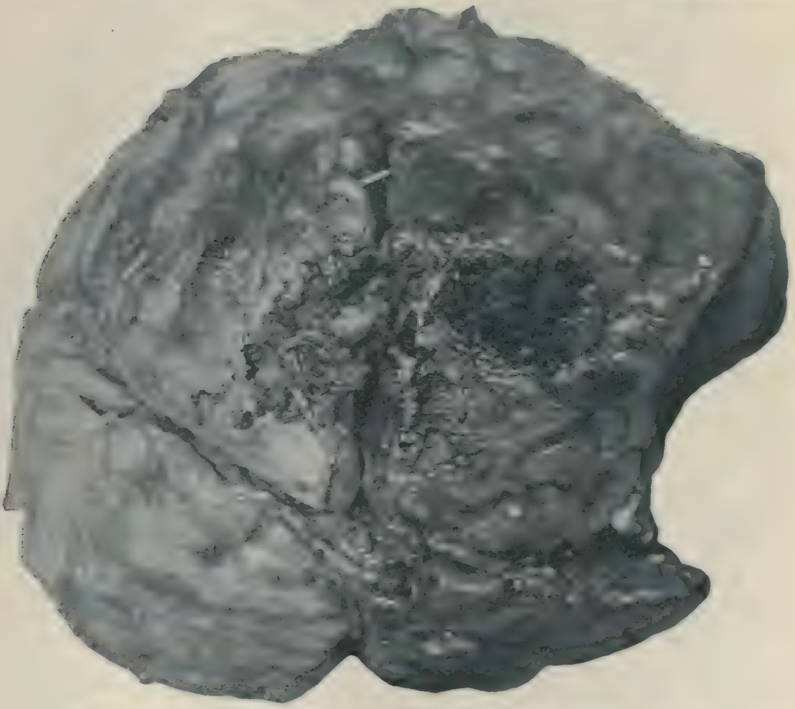


FIG. 4. Case 2. Showing areas of contusion—and hemorrhage into cortex.

The left side of the face was distinctly paretic in both the upper and lower distributions of the facial nerve. She closed the eyelids of the left side but with less force than on the right. The tongue was protruded straight. There was left hemianopsia. Sensation to touch and pain was diminished on the whole left side of the body, including the face. The left arm and leg seemed to be completely paralyzed. The triceps and biceps tendon reflexes were very prompt on both sides but more so on the left.

The left arm and leg were flaccid. The patellar reflex was prompt on each side and a little more so on the left. The Achilles jerk was not obtained on either side. There was no ankle clonus and the Babinski phenomenon was uncertain on the left side and probably absent on the right. At a previous examination it was present on the left side.

Two days after admission the patient was operated upon, a diagnosis of brain tumor having been made in the absence of any history of injury. The skull was opened on the right side over the motor area. No tumor was found but when the dura was opened large quantities of fluid somewhat blood tinged escaped. The hemorrhage was readily controlled and the dura was allowed to fall into place without suturing. The patient died the following day.

In the brain there was found on the right side in the region of the frontal lobe and especially over the face center an extensive sub-pial hemorrrage which extended down to the basal surface. There was also an area of hemorrhagic infiltration in the region of the arm center. A hemorrhagic area in the temporal lobe on the basal surface was observed. In addition to this a sub-dural hemorrhage in the region of the parietal lobe on the right side was seen and in the posterior part of the left cerebellar hemisphere there was also an area showing softening from bruising or contusion.

Microscopically in the precentral region there was seen a hemorrhage which at first sight appeared to be a hemorrhagic area in the brain cortex but which proved to be an accumulation of blood beneath the pia which pushed the brain matter down beneath it. Just beneath the pial hemorrhage the cortical tissue showed very little damage. The main lesion consisted of a hemorrhage into the cortex at the site of the contusion. In the tissue adjoining the area of contusion there was some rarification of the tissue and space formation. In the frontal region there was a hemorrhage between the pia and the arachnoid which pressed down on the cortex. In most places the cortex was perfectly normal but in one place the hemorrhage had extended into the cortex. The pia, at the seat of the hemorrhage was vascular, and the walls of the vessel were thicker and the lumen larger than normal. The pia itself was thickened and stained amorphously. There was not found any round cell infiltration.

Adjoining the hemorrhage into the cortex were smaller hemorrhages and space formation. In one section an enormous pial hemorrhage did not materially injure the cortex and in only a small area was there seen any disintegration of the cortex from the hemorrhage. There was in the pia very extensive hemorrhagic extravasation which occurred independent of any implication of the cortex.

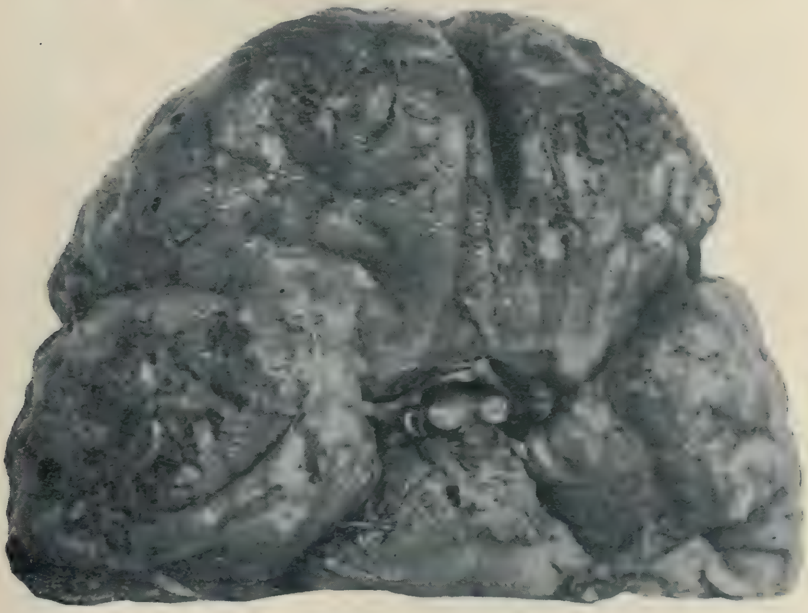


FIG. 5. Case 2. Showing areas of contusion.

In the areas of contusion the brain substance was infiltrated with red blood cells to such an extent as to entirely mask any of the normal appearance of the brain. Adjoining this before reaching normal brain tissue were seen many microscopic hemorrhages. Here the ganglion cells could be seen well stained and intact.

There was no evidence of any inflammatory process or reaction. The sections stained with thionin showed no degeneration of the nerve cells in the cortex.

CASE 2. F. S., aged 53, was admitted to the Philadelphia Polyclinic Hospital on January 24, 1913. He was brought into the accident ward in an unconscious condition having been knocked down in a fight, his head as he fell striking the cement floor of an abattoir where he worked. There was no external evidence of injury with the exception of some contusion over the left eye and in the occipital regions. He was not entirely comatose upon admission but did not make replies to any questions. He was restless and red faced. The pupils were contracted and reacted to light. As far as could be ascertained there did not seem to be any paralysis upon admission.

On January 26 two days later he was still unconscious and there was incontinence of urine and feces. Both knee jerks were increased and there was a Babinski phenomenon on the left. On this date two days after the acident there was observed twitching of the right side of the face which became almost constant and continued until the patient died five days later. On the twenty-eighth two days after the convulsions of the face began, the right arm became the seat of clonic convulsions and on the following day the right leg also became involved. He died seven days after accident without regaining consciousness.

At the autopsy a fracture of the base of the skull extending from the occipital region to the frontal region on the left side was found. The whole brain was very badly bruised. In the left precentral region involving the second and third frontal convolution in their posterior parts there was an area of hemorrhagic softening measuring 6 cm. by 3 cm. Extensive sub-pial hemorrhage surrounded this area and extended forward and downward to the basal surface. There was a sub-dural hemorrhage in this region also. On the basal surface of the brain could be found areas of softening or contusion of the brain tissue occupying the region of the anterior temporal lobe and the anterior portion of the frontal lobe. Surrounding these areas was found extensive hemorrhage into the pia to a greater or less extent.

Microscopically studies were made of the contused areas. The pia arachnoid was in places alone involved, the hemorrhage separating the pia from the arachnoid. Beneath these places the cortex was intact, showing nothing abnormal with the nuclear stain and no destruction or alteration of the cells when stained with thionin.

In the areas of contusion the brain substance was infiltrated with blood and here there could be seen no traces of the normal brain tissue. Adjoining these areas minute hemorrhages were seen in the cortical layer for a short distance in transition to healthy brain tissue. The cells in these localities showed no change though perhaps they took the stain less intensely than the normal. Just adjoining the extensive hemorrhagic infiltration of the cortex in one place the cells stained poorly by the thionin stain and a few very poorly or not at all. In the pia there was no evidence of proliferation or exudation of round cells, nor in the cortex at the seat of contusion was there any round cell infiltration.

CASE 3. W. B.. aged 46, was admitted to the Philadelphia Polyclinic Hospital on December 16, 1913. He was in a somewhat dazed condition upon admission and complained of pain in the head, having received a contused wound in the right parieto-occipital region. Twenty-four hours later he had a general convulsion. Upon examination two hours after the convulsion his condition was as follows. He was confused and had difficulty in expressing himself. Further examination revealed the fact that he was aphasic. He was unable to understand spoken language or writing and was unable also to express himself, although there was no anarthria. There was a tendency to divergence of the right eye. The pupils were very small but reacted promptly and the rotation of the eyeballs was good. There was possibly a slight twitching of the right external rectus. When examined by Dr. Peter the next day, the eye grounds were practically negative.

On the following day an examination showed in addition to the aphasia some apraxia. For example repeatedly when asked to close his eyes he would show his teeth or he would make a fist.

An X-ray examination showed a possible fracture in the right occipital region. There was a gradual improvement in his condition, including disappearance of the aphasia and apraxia which persisted for about a week. His chief complaint then consisted of pain in the occipital region and over both scapulas. Mentally he was very slow and answered questions in monosyllables.

At the end of five weeks he was discharged cured. (A week later he committed suicide by inhalation of illuminating gas.)

This case was one in my opinion of contusion or perhaps laceration of the temporal region on the left side causing apraxia and aphasia. The hemorrhage into the cortex must surely have been absorbed allowing thus the function of the brain tissue to be restored, the pressure being relieved by absorption of the hemorrhage.

It would seem to me in studying the cases of so-called concussion, in which the symptoms persist for several days and in which even weeks pass by before the patient regains the normal state that the condition is best explained on the basis of a contusion or actual injury to the brain substance itself. The brain becomes bruised as it is thrown against the bony structure of the cranium. The difference between concussion and laceration is one largely of degree, there being no sharp line between concussion and contusion on the one hand or contusion and laceration on the other.

LITERATURE

1. Kocher. Nothnagel's System, Wien, 1901.
2. Keen. In DaCosta's Modern Surgery, 1914.
3. Koch and Filehne. In Cushing. Keen's System of Surgery, Vol. 3, p. 186.
4. Witkowski. In Cushing.
5. Phelps. New York Med. Jour., 1893, p. 29.
6. Page. Brain, 1902, Vol. 25, p. 1.

Reprinted from The Journal of the American Medical Association
Nov. 7, 1914, Vol. LXIII, pp. 1662-1666

TUMOR OF THE CRUS CEREBRI *

JOHN H. W. RHEIN, M.D.
Professor of Diseases of the Mind and Nervous System at the Philadelphia Polyclinic and College of Graduates in Medicine;
Neurologist to the Howard Hospital; Physician to the Philadelphia Home for Incurables
PHILADELPHIA

Tumors of the crus cerebri are rare. In the collection of the brains in the neurologic laboratory at the University of Pennsylvania there is only one instance, a case of Dr. Spiller's, to which reference will be made later. There is also a second case of glioma of the pons extending to the crus. A careful search of the literature has been made and only a few cases have been reported. Under these circumstances it seems justifiable to report a single case.

M. S., aged 12, colored, was admitted to my service at the Philadelphia Polyclinic, Dec. 1, 1912. The family history was negative. There was no history of tuberculosis, syphilis or mental disease. Both parents and one brother were well. Her birth had been normal and the child developed normally, physically and mentally. As an infant she suffered from measles, chicken-pox and whooping-cough.

Two years previous to admission she had suffered from an attack of typhoid fever and in the previous summer had had a second attack which her parents stated was associated with malaria.

Two years previously she partly lost the use of her right arm and hand, and after an attack of typhoid malaria eighteen months later the right leg became weak. Two months ago there developed pains over the left eye which radiated to the left ear, and ptosis of the left eye. During the two weeks before admission to the hospital she had vomited every morning immediately after breakfast and had convulsions of the right side of the body lasting two or three minutes and recurring several times during the day. She was stuporous during the attack, but regained consciousness immediately after the twitching ceased. There

---

*** Read before the Section on Nervous and Mental Diseases at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.**

has been for four months difficulty with the rectal and vesical sphincter, the latter consisting of difficulty in emptying the bladder.

On examination the patient was found to be a poorly nourished colored girl. The physical examination of the heart and lungs proved to be normal. The liver and spleen were not enlarged and there were no palpable masses in the abdomen.

There was paralysis of the right side of the face in the lower distribution of the facial nerve. The patient moved the left side of the face and both sides of the forehead well and closed both eyes equally well. There was some rigidity of the neck when rotary or to-and-fro movements of the head were made. The pupils did not react to light or in accommodation. In making an effort to follow an object to the right the right eyeball did not move beyond the median line and the left eyeball moved only a slight distance to the right. In following an object to the left the right eyeball moved to the inner canthus and the left eyeball remained motionless. In following an object upward neither eyeball moved at all. In following an object downward the right eyeball moved only slightly and the left eyeball not at all.

The tongue protruded markedly to the right. The patient winced when the face was scratched with a sharp point on both sides. The finger to nose test showed marked ataxia on the right side and slight on the left.

In the right arm there was marked contracture of the biceps muscle which caused flexion of the forearm on the arm. She could not voluntarily extend the forearm. The grasp of the right hand was almost nil and was feeble on the left side. Rotary movements of the right arm could scarcely be made at all. The left arm was feeble but all movements were possible. There was no diadokokinesia. In walking she dragged the left leg, although both legs appeared to be weak.

Both knee-jerks were exaggerated and equal. There was no ankle-clonus, but the Babinski sign was present on both sides. The toes on both sides were hyperextended, especially on the right. Weakness of the legs prevented a satisfactory test for diadokokinesia. An examination of the eye by Dr. Zentmayer showed intense swelling of the nerve-head and surrounding retina with a few scattered hemorrhages. Later the pupils became unequal, the right being smaller than the left and the left eyeball protruded more than the right. A diagnosis of tumor of the upper part of the pons was made.

Necropsy.—The left crus cerebri was very much enlarged and extended to the right of the median line. On section it was found that the enlargement was due to a caseous tumor which extended to the upper limit of the pons below and the basal ganglia above. The internal capsule was somewhat implicated in its anterior limb and the optic thalamus was also apparently involved in its anterior third. The focus in the pons extended to the opposite side to a slight degree. There was also a small focus of circumscribed caseation in the white matter of the frontal convolution on the left side situated half an inch to the outside of the caudate nucleus. On microscopic section the tumor proved to be a tubercle.

Summary.—In a girl of 12 there developed gradual loss of power in the right side of the face, the right arm and leg, left sided ptosis and convulsive seizures confined to the right face, arm and leg. Later paralysis of associated movements upward and downward, and laterally, and finally weakness of the left arm developed. The symptoms were caused by a tubercle of the left crus cerebri which invaded the upper part of the pons, affecting the nuclei of the third nerve and extending upward into the basal ganglia. The paralysis of associated movements upward and downward and laterally indicated a lesion of the pons in the neighborhood of the oculomotor nuclei, and a lesion of the posterior longitudinal bundle.

To the diagnostic value of these symptoms Spiller has already called attention. It was the presence of the paralysis of associated movements which led to a diagnosis of tumor of the pons in this case. The extent of the tumor was not suspected during life. Indeed, I know of no way to differentiate between tumors of the crus and of the upper part of the pons.

It is not a surprising observation to find the two associated, to which the cases of Touche,[1] Sharkey,[2] Mann and Delépine[3] and Hun[4] will testify.

Obviously the more caudal the lesion the more apparent it becomes that the lesion affects more than the crus.

When the lesion extends upward and involves the external geniculate body or implicates the optic tracts, there intervenes hemianopsia and hemiopic pupillary inaction. Motor ataxia or sensory disturbances indicate extension of the lesion to the tegmentum.

In a review of the literature only seventeen cases of tumor of the crus cerebri were found in which the diagnosis was verified by necropsy.

Sanz[5] reported a case of a child of 8. The right arm and leg were paralyzed and contractured and there was left ophthalmoplegia. To these symptoms were added delirium, fever and later right ophthalmoplegia and bulbar disturbances. The necropsy revealed a single tubercle in the left cerebral peduncle. Both peduncles were softened as well as the pons and medulla. There was some meningeal reaction.

---

[1] Touche: Neurol. Centralbl., 1902, xxi, 42.
[2] Sharkey: Brain, London, 1894, xvii, 238.
[3] Mann and Delépine: Brain, 1898, No. 21, p. 475.
[4] Hun: Jour. Nerv. and Ment. Dis., 1887, xiv, 624.
[5] Sanz: Rev. Espagne Med. y Cirur. Práct., 1908, lxx, 39.

Hunt[6] described a case of a boy of 17. A month after an injury to the head there developed strabismus and ptosis of the right eye followed by weakness and stiffness of the left arm and leg. There was somnolence, vertigo and inordinate laughter on slight provocation and sometimes spontaneous. There was no headache, nausea or vomiting. There was complete ptosis of the right eye, partial of the left, divergent strabismus on both sides, and paralysis of the upward and downward and inward movements of the right eye. The inward and downward movements of the left eye were also limited in their excursion. Some nystagmoid movements were present. The pupils reacted normally but sluggishly. The disks were congested and the upper margin was hazy.

The left side of the face, arm and leg were paralyzed and spastic. Ankle-clonus and the Babinski phenomenon were present but no objective sensory disturbances. The skin reflexes were absent on the left side. There was uncontrollable and involuntary laughter from time to time. The tongue deviated toward the left. Later there were involuntary urination and defecation. The motor portion of the fifth nerve was paralyzed on one side, the jaw deviating toward the right side.

At the necropsy was found a tumor extending from the right crus cerebri. It was a firm, nodular, grayish-white mass in appearance filling up the interval between the cerebral peduncles. The right third nerve was compressed and atrophic. There was also a cyst springing from the floor of the fourth ventricle measuring 1¼ inches by ⅝ inch. It dipped down into the substance of the pons and passed beneath the aqueduct and appeared on the inferior aspect of the right crus cerebri, where it became adherent to the tumor. Degenerative changes were found in the medulla and pons. The tumor in this case proved to be a glioma.

---

[6] Hunt: Am. Jour. Med. Sc., 1904, cxxvii, 514.

Henoch[7] reported a case of tuberculosis in a child of 3 who died twelve weeks after the onset of the symptoms. There was first tremor of the left hand, arm and leg. The fingers were held in a flexed position and there was right-sided ptosis, dilatation of the right pupil and external strabismus. Later there was ptosis, mydriasis and strabismus of the left eye. Death ensued as a result of measles and bronchopneumonia. A tumor the size of a cherry was found on the right cerebral peduncle extending into the third ventricle. The right third nerve was flattened, thin and discolored. There was also a tuberculous area in the apex of the left lung.

Garnier[8] described a case in a man of 65. The head and eyes deviated to the right and there was complete right-sided hemiplegia with slight rigidity. The temperature was lower on the left side. The patient died one hour and a half after admission.

A small ovoid tumor was observed on the right cerebral peduncle, which proved to be a lipoma. It was adherent to the base of the cerebral peduncle but it did not look as if it were the cause of the compression of that organ, the hemiplegia being probably due to a cerebellar hemorrhage which was also present. He stated that only thirty instances in the literature were found of lipoma in the central nervous system. These tumors originate in the meninges or their vicinity.

D'Astros and Hawthorn[9] cited a case of tubercle of the cerebral peduncle in a girl of 21 months. The symptoms began at 10 months of age after some digestive troubles. There developed trembling of the left hand and arm, some photophobia, right oculomotor paralysis characterized by ptosis, external strabismus with immobility of the eyeball and partial mydriasis with sluggish reaction of the pupil. There was paresis of the left side of the face but no involvement of the fifth nerve.

A conspicuous symptom was the rhythmic tremor of the forearm consisting of an oscillatory movement from side to side and forward and backward. The left leg was paralyzed and spastic. There was also some alternating flexion and extension of the muscles of the left leg. There was no Babinski sign. The patellar reflexes were exaggerated on both sides.

---

[7] Henoch: Vorlesungen über Kinderkrankheiten, Berlin, 1903, Ed. ii, p. 270.

[8] Garnier: Rev. méd. de l'est., 1902, xxxiv, 590.

[9] D'Astros and Hawthorn: Rev. Neurol., 1902, x, 377.

The patient died about twelve months after the onset of the disease. There was besides tuberculosis of both lungs a caseous tubercle, the size of a cherry about 2.5 mm. below the protuberantia and extending forward to the wall of the third ventricle. It occupied almost the entire upper portion of the right peduncle. He also cites the case of Raviard and Asset in which there was a tremor on the right side with left-sided ocular motor paralysis due to the large peduncular tubercle.

Blocq and Marinesco[10] described a case exhibiting a tremor like paralysis agitans which was confined to one side, in which there was a tumor of the cerebral peduncle, occurring in a man of 42. The first symptoms consisted of swelling and stiffness of the left side of the face, the eyes and tips of the fingers of the left hand. This rigidity progressed and at the end of a year and a half, tremor began. On examination the expression was Parkinsonian. There was a rhythmic tremor of the left hand and leg controlled for a short time by the will. The body was bent forward in walking and there was a slight tendency to stumble to the left. Transitory diplopia was noted. The left patellar reflex was exaggerated. No sensory change except subjective tickling of the left hand was noted. A diagnosis of unilateral Parkinson's disease was made. At the necropsy there was a tuberculous lesion found in the thorax and lumbar vertebrae. The right cerebral peduncle was enlarged and the seat of a tumor the size of a nut. There were no degenerative lesions.

Pepper and Packard[11] described a case of glioma of the right crus cerebri in a boy 4 years of age. There was left-sided facial and corporal hemiplegia. The tongue protruded to the left and there was transient involvement of the left extrinsic ocular muscles. There was a marked contracture of the arm but none of the leg and there were no headaches, sensory disturbances, hemianopsia or paralysis of the masticatory muscles. Death followed convulsions. At the necropsy there was a tumor the size of a hazelnut seated at the outer and under surface of the right crus cerebri. The microscopic examination showed it to be a glioma.

---

[10] Blocq and Marinesco: Compt. rend. Soc. de biol., 1893, v, 105.
[11] Pepper and Packard: Am. Jour Med. Sc., 1890, cxix, 263.

Benedikt[12] cited a case of a boy of 4 who suffered from headache for six weeks before applying for treatment, at which time there was right-sided ptosis and oculomotor palsy of the same side. The head was turned toward the left and the chin toward the right. There was a tremor of the left hand and leg, which were paretic. The muscles of the lower lip were paralyzed, deviating toward the left. At the necropsy there were a number of tubercles found in the left frontal lobe, in the marginal convolutions, in the cuneus, and in the left cerebellar hemisphere. Finally a tubercle the size of a pigeon's egg occupied the inferior surface of the right cerebral peduncle. This tumor destroyed the right oculomotor nerve. He also cited a second case in a man with pulmonary tuberculosis presenting crossed oculomotor paralysis and hemiparesis with tremor of the paralyzed members.

Kraft-Ebing[13] described a case in a woman of 41 with tubercle of the right cerebral peduncle. The first symptoms consisted of heaviness in the legs associated with habitual headaches, right-sided tinnitus aurium, vertigo and weakness in the right arm with drawing pains in the right arm and leg. Diplopia and bilateral ptosis developed later and there was paralysis of the superior rectus muscles with pronounced right-sided papillitis. The deep reflexes were enormously exaggerated on the left side and there was right-sided hyperalgesia. There was persistent left hemiataxia. Death ensued at the end of three months. The necropsy revealed a tubercle 13 by 11 mm. in size occupying the entire tegmental nucleus. There was general tuberculosis.

Mendel[14] presented a specimen of tubercle from the brain of a child of 4 years and 9 months of age. The first symptoms consisted of an intention tremor of the right arm which later was followed by a weakness of the right leg, left-sided ptosis and paralysis of the right externus with dilation of the left pupil, which was twice as large as the right. The right side of the mouth appeared paralyzed, the face being drawn toward the left. The tongue protruded toward the right and the right neck-muscles were paralyzed. The right arm also was paretic. Sensation was not implicated. The temperature of the right arm and leg was, however, reduced. A large tubercle measuring 2 by 1.25 cm. was found in the left peduncle. The red tegmental nucleus of the superior peduncle seemed to be completely destroyed. Anteriorly the tumor extended to the left corpus subthalamus. There was some circumscribed meningitis in the interpeduncular space, which would explain the right-sided ocular motor paralysis.

---

[12] Benedikt: Bull. Med., 1889, iii, 547.

[13] Kraft-Ebing: Wien. klin. Wchnschr., 1889, ii, 896.

[14] Mendel: Berl. klin. Wchnschr., 1885, xx, 468.

Ramey[15] described a case of alternating paralysis of peduncular origin in a man of 52. Following occasional transitory sensation of tingling in the right foot, there followed a gradual paralysis of the right arm, leg and face. The knee-jerks were exaggerated on both sides. There was some diminution of sensation of the right side and the temperature was much lower than that on the other side. There was paralysis of the left third nerve, including the iris and the ciliary muscles. The right eye showed paralysis of the inferior, superior and external muscles. There was considerable contraction of the fields of vision. The necropsy revealed the presence of a miliary tuberculosis of the lungs. The left peduncle was larger than the right and was the seat of a tumor consisting of an agglomeration of eight to ten small yellowish masses.

Proust[16] cited a case of tumor of the right cerebral peduncle in a man of 32 who two years prior to coming under observation developed headache, vertigo and deafness. Three months previously there gradually developed an incomplete hemiplegia of the left side involving the face. The left forehead appeared much less wrinkled than the right. The left eyelid was opened less easily than the right and protruded more than the right. The pupil was dilated and there was some strabismus. There was some slight diminution of sensation of the left side. He emitted from time to time piercing cries. There was no vomiting, loss of consciousness or convulsions. At the necropsy a tumor the size of an egg was found occupying the right cerebral peduncle. There was distention of the right lateral ventricle. The peduncle was completely flattened.

---

[15] Ramey: Rev. de méd., 1885, v, 489.
[16] Proust: Progrès méd., 1882, x, 187.

Ria[17] added a case of a glioma of the left cerebral peduncle in a man of 33. The first symptoms consisted of a slight weakness of the right hand and a month later there developed weakness of the right leg and in still another month difficulty in speech. Later there was ptosis of the left upper eyelid. Tactile, pain and heat sensations were only a trifle below normal. The left pupil was dilated and there was external strabismus. At the necropsy a tumor the size of a nut was found in the left peduncle which extended to the optic thalamus and affected the corpus striatum. It also involved the left large ventricular ganglia. Under the microscope the tumor proved to be a glioma.

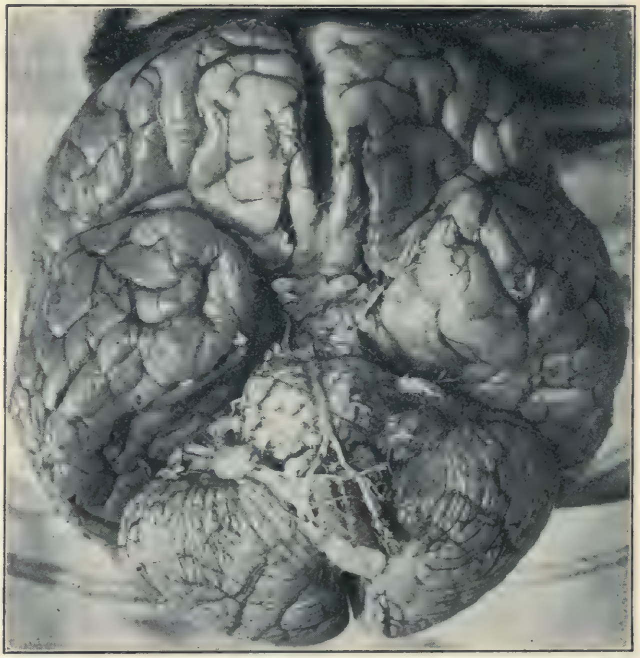
Archambault[18] presented a case of complete paralysis of the right ocular motor nerve and incomplete left hemiplegia in a child of 3 years of age. At the necropsy there was a tuberculous tumor the size of a nut in the right cerebral peduncle a little in front of the third ocular motor nerve. There was also a tuberculous lesion of the lungs.

Bouveret and Chapotot[19] described a case of a woman of 22 who awoke with weakness of the left side which was shortly followed by oculomotor paralysis and frequent headaches which were associated with vomiting and vertigo. Two months later there was partial paralysis of the left side of the face and complete left hemiplegia. Sensation to heat and cold was lowered on the left side. A slight ptosis of the right side was observed, but all the muscles of the left eye were more or less involved. The left pupil was larger than the right. There was incipient optic neuritis. About a month later there were left-sided convulsions and about two weeks after this there was paralysis of the right third nerve and dilatation of the right pupil. Choreiform movements followed shortly after this. The patient died six months after the onset of the symptoms. At the necropsy there was discovered tuberculosis of the lungs. A cherry-sized caseous mass was found at the junction between the gray and white substance at the base of the second frontal convolution of the left side. Both peduncles were the seat of a tuberculous tumor. On the right side the tumor projected toward the occipital horn of the lateral ventricle and extended under the aqueduct of Sylvius, where it destroyed the gray matter.

---

[17] Ria: Ann. clin. d'Osped. Incur., 1885, v, 82.
[18] Archambault: Progrès méd., 1877, v, 718.
[19] Bouveret and Chapotot: Rev. de méd., 1892, xii, 728.

Beck[20] reported a case of hard round-cell sarcoma in the left crus in a male. The first symptoms were attacks in which he would feel weak and experienced a sudden sensation of shock which caused the head and upper part of the body to bend forward and which was associated with vertigo. He also suffered from severe frontal headache. On examination there was found diplopia, the right pupil was smaller than the left, which did not react at all to light, and there was convergent strabismus in the right eye, the left eye remaining stationary. The ophthalmoscope showed a fulness of the veins. The right facial nerve was slightly paralyzed. The tongue protruded straight and there was no speech disturbance. The movements of the right arm and leg were much slower than on the left and the patellar and Achilles reflexes were exaggerated. Two months later there developed complete ptosis of the left side and a slight ptosis of the right.



Enlargement of left crus cerebri.

---

[20] Beck: Virchows Arch. f. path. Anat., 1883, xciv, 378.

The left ocular muscles were paralyzed with the exception of the rectus externus. All symptoms became aggravated until death about six months after applying for treatment. At the necropsy a spherical tumor was found measuring 1.5 by 2 cm. situated in the left cerebral peduncle and reaching as far as the ganglia. The oculomotor nerves were flattened by the pressure from the tumor.

Spiller[21] described a case in a man of 48 years of age who had paralysis of upward associated ocular movements with loss of convergence. The downward movement was difficult, while the lateral movements were intact. The speech was bulbar in character and there was dysphagia associated with constant dribbling of saliva. He complained of headache, extreme vertigo and uncontrollable spells of laughter. Later all associated ocular movements became impaired and there was slight ataxia of the arms.

At the necropsy there was found a tumor of the right cerebral peduncle which measured 2 by 5 mm. and consisted of a sarcoma, possibly endothelioma. The lowest portion of the tumor was at a point where the fibers of the oculomotor nerves leave the nuclei in large bundles and it extended toward the aqueduct almost to the lowest part of the oculomotor nucleus. A few fibers of the right oculomotor nerves passed through it. Posteriorly the tumor extended a little beyond the aqueduct of Sylvius.

The characteristic symptom complex of tumor of the crus cerebri is said to consist of a hemiplegia on the opposite side to the lesion associated with oculomotor palsy on the same side. This has been termed hemiplegia alterans superior. But in fourteen of eighteen cases collected from the literature and including my own, the hemiplegia was associated with oculomotor palsy on both sides in nine instances (Hunt, Henoch, Kraft-Ebing, Mendel, Rhein, Ramey, Bouveret and Chapotot, Beck, Benedikt) and only in five cases was the oculomotor paralysis unilateral (Sanz, d'Astros and Hawthorn, Benedikt, Ria, Archambault). The close proximity of the two nuclei of the third nerve explains the frequency of the involvement of the oculomotor nerve on the opposite side. In rare instances the paralysis and oculomotor palsy may be on the same side. Farnell and Burnett described such a case but there was no necropsy. In Pepper and

---

[21] Spiller: Jour. Nerv. and Men. Dis., 1905, xxxii, 497.

Packard's case with necropsy the hemiplegia and third nerve palsy were on the same side.

The hemiplegia is gradual in onset and is usually associated with spasticity.

In some cases a tremor or choreiform movements are present. In the cases cited in this paper this symptom was present in eight instances (Henoch, d'Astros and Hawthorn, Bouveret and Chapotot, Raviard and Asset, Blocq and Marinesco, Benedikt (two), Mendel). In d'Astros and Hawthorn's case the tremor consisted of a rhythmic tremor of the forearm in which the movements were from side to side and forward and backward. In Bouveret and Chapotot's case the tremor was choreiform in character. In the case described by Blocq and Marinesco the tremor was like that of paralysis agitans. Also in the two cases cited by Benedikt there was a tremor of the paralyzed limbs. In Mendel's case an intentional tremor of the right arm was the first symptom. The tremor must be looked on as an irritating symptom due to pressure on the pyramidal tracts.

Hemiataxia has been noted, as in the cases of Kraft-Ebing and Spiller. Ataxia was present in both arms in the case cited by myself in this paper.

In rare instances palsy of the opposite limbs may be observed, when according to Oppenheim[22] dysphagia and dysarthria may occur.

Sensory disturbances are present in some cases consisting of contralateral hemianesthesia. Parasthesia and hemihyperesthesia have occurred.

Reduction of the bodily temperature on the paralyzed side has been noted in some cases (Garnier, Mendel, Ramey). This possibly may be referred to vasomotor disturbances due to implication of the substantia nigra. Inordinate laughter occurred in two cases, namely, those of Spiller and Hunt. Of the eighteen cases, twelve were tuberculous tumors (Sanz, Henoch, d'Astros and Hawthorn, Blocq and Marinesco, Benedikt (two), Kraft-Ebing, Mendel, Ramey, Archambault, Bouveret and Chapotot, Rhein). In one the tumor was a lipoma (Garnier), in three cases glioma (Pepper and Packard, Ria, Hunt), and finally, in two cases the tumor was a sarcoma (Beck, Spiller).

1732 Pine Street.

---

[22] Oppenheim: Spec. Path. u. Therap., Nothnagel, Wien., 1896, ix, 125.

[Reprinted from The Journal of Nervous and Mental Disease, Vol. 41, No. 6, June, 1914.]

ANEURISMS OF THE VESSELS OF THE BRAIN[1]

By John H. W. Rhein, M.D.

PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM AT THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE; NEUROLOGIST TO THE HOWARD HOSPITAL, ETC.

A study of the literature reveals the fact that aneurisms of the cerebral vessels are not uncommon, the first contribution to the literature having been made by Morgagni,[2] as long ago as the early part of the eighteenth century, since which time many cases have been recorded.

The literature is so rich that one feels like apologizing for reporting a single case. At the same time the one, a report of which follows, presents some features of interest; chiefly as illustrating the absence of symptoms recognizable as being due to the aneurism and as showing that paralyses which are met with in some of the cases may be coincidental rather than related directly to the aneurism itself.

B. R., age 57, was admitted to the Philadelphia Home for Incurables on June 11, 1908, and died December 10, 1911. The chief complaint was loss of power in both legs.

Father died of paralysis, otherwise the family history is entirely negative.

There is nothing in the previous history bearing on his present illness. He denied syphilitic infection.

He stated that in 1906 he had a "stroke." While walking, he turned to the left suddenly but did not fall or lose consciousness. There was immediately slight loss of power of the left arm and leg. Previous to this attack he stated there was a slight lameness of the left leg, which came on gradually.

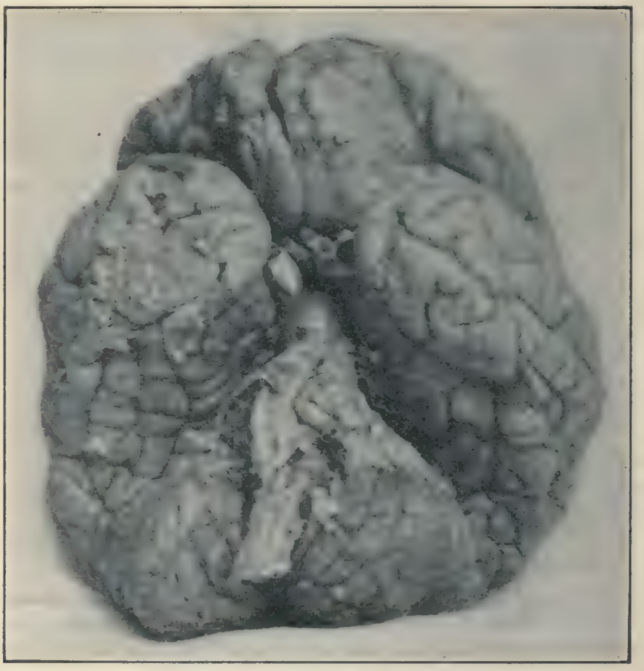
Upon examination there was loss of power in both legs. He was able to walk but the gait was slightly spastic. Apparently there was no loss of power in the arms at the time of the first examination. There were no sensory disturbances of either the arms or legs. The knee jerks were increased, ankle clonus was present on both sides and the Babinski phenomenon was present. There was no involvement of the sphincters at this time.

---

[1] From the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania, and the Philadelphia Home for Incurables. Read before the Philadelphia Neurological Society, March 27, 1914.

The weakness of the leg progressed so that two months before his death he was unable to walk even with crutches. The arms became slightly spastic and there was some loss of power on both sides. In the last few months of his life the bladder sphincter was at times incontinent. There was no localized wasting of the muscles. Shortly before death he was asthmatic and dyspneic and he complained of pain over the gall bladder.

At the autopsy the base of the skull was somewhat pushed out of shape. The posterior clinoid processes were pushed forward and the anterior posterior diameter of the sella turcica was shortened.



Aneurism of the Basilar Artery.

At the anterior portion of the basilar artery an aneurism was found the size of a small pigeon's egg. The aneurism was situated at that point where the basilar artery divides into the posterior cerebral arteries. The posterior communicating arteries were very small and threadlike in appearance. The diameter of both internal carotids was abnormally large.

The right kidney was much enlarged and was the seat of a tumor in its upper portion. The left lung was infiltrated with nodular masses varying in size from a hickory nut to a hen's egg. These tumors could be readily enucleated. The plura was thickly studded with small nodules, the spleen was somewhat enlarged and the gall bladder was distended with pus and contained several gall stones. The left kidney was slightly enlarged but was not the seat of any growth. Microscopic examination of the tumors was made by Professor Allen J. Smith, who reported that the tumor was a hypernephroma.

Sections from the paracentral regions showed marked arterial change in the vessels of the cortex, consisting chiefly of perivascular round cell infiltration and thickened arterial walls. The pia in these regions was thickened, fibrous and infiltrated markedly with round cells. The blood vessel walls were thickened and some fresh hemorrhages were noted in the pia. In the main the pyramidal cells and Betz cells were not diseased.

Microscopic studies were also made of the pons, medulla oblongata, and the spinal cord.

The pons half an inch below the seat of the aneurism showed areas of round cell infiltration which were situated in the neighborhood of the junction of the lateral and median lemnisci.

The motor tracts here were not degenerated, showing that the paralysis was due to some lesion lower down than the peduncle, or in the pons at the level of the aneurism, and hence not due to the pressure of the tumor, as was at first suggested.

In the medulla oblongata the right lemniscus in its posterior part was degenerated; also the fibers in the lower part of the olive. In the pyramids there was marked round-cell infiltration, and the walls of the blood vessels were thickened. This was more marked on the left side. Both pyramids were degenerated, the left diffusely and the right in its outer angle markedly so, sharply defined here and diffusely so in the remainder of the right pyramid. This was probably due to the round cell infiltration in the pyramids.

The pia of the medulla was intensely infiltrated with round cells, and the blood vessel walls were thickened.

The pia of the spinal cord was the seat of a round cell infiltration and some fresh hemorrhages.

The crossed pyramidal tracts on both sides were degenerated and this was more marked on the left side. The degeneration extended from the cervical to the lumbar regions.

The paraplegia, and the spasticity and weakness of the arms were due to the disease of the crossed pyramidal tracts resulting from foci of round cell infiltration somewhere in the lower part of the pons or in the medulla oblongata and of specific origin probably. These symptoms were independent of the aneurism, which caused no symptoms during life and was recognized for the first time at the autopsy.

Let us first discuss briefly the symptomatology of cerebral aneurisms in general with special emphasis upon the symptoms of aneurisms of the basilar artery.

It must be borne in mind that a number of the cases of cerebral aneurism present no symptoms being found accidentally at autopsy. A second group of cases present apoplectic symptoms as the first symptoms. In a third group of cases we have symptoms of apoplexy, which have been preceded by symptoms of cerebral tumor. Finally, in the fourth group of cases, there may be symptoms of cerebral tumor only.

In 555 cases collected by Beadles,[3] 92 cases presented no symptoms. In about 20 per cent. of the cases, the symptoms suggested those of tumor and these preceded fatal apoplectic stroke. In over 16 per cent. of the cases the symptoms were those of a cerebral tumor, and in 46 per cent. of the cases the symptoms were those of apoplexy.

The symptoms of cerebral aneurisms in general are as follows: Headache, vertigo, vomiting, mental disturbances, loss of memory, deafness, disturbances of speech and respiration, more rarely affections of sight, convulsions, hemiplegia, paraplegia, oculo-motor palsies, still more rarely involvement of the third, fourth, and fifth, as well as the olfactory nerves. Bitemporal hemianopsia has been recorded in a few cases (Mitchell,[4] Bramwell[5]).

As has been stated a great many cases show no symptoms at all, especially those in which the middle cerebral and basilar arteries are the seat of an aneurism.

The pressure symptoms vary according to the size, situation, and rapidity of growth.

The situation of the aneurisms has more bearing upon the symptomatology than the size of the tumor. Processes that may accompany the aneurisms, such as meningitis, hemorrhage, thrombosis and areas of softening, modify the symptoms.

Aphasia is more likely to occur in aneurism of the middle cerebral artery.

Hemiplegia occurs in aneurisms of the middle cerebral and basilar arteries, as well as the posterior cerebral artery.

It is said that paraplegia occurs only in aneurisms of the basilar artery.

Aneurisms of the posterior communicating artery cause oculomotor palsies and pressure upon the optic tracts.

The optic and olfactory nerves may be paralyzed in aneurisms of the anterior cerebral artery. The cranial nerves are rarely implicated except the facial nerve. The involvement of the optic nerve is rare.

Mental disturbances, which occur frequently in the history of cerebral aneurisms, are probably an accidental association but may be due in part to vascular degeneration of the cerebral arteries.

Opinions differ as to the value of murmurs in the diagnosis of cerebral aneurisms. Epron[6] considers the presence of a murmur as pathognomonic and Bruns[7] while recognizing the value of a bruit called attention to the fact that it may be also caused by chlorosis, sinus disease and thrombosis. On the contrary Oppenheim[8] did not believe that murmurs were to be looked upon as a safe sign and Beadles in discussing the value of murmurs claims that out of 555 cases collected there were only two in which a murmur proved to be due to an aneurism. Gowers[9] believed the presence of murmurs to be a rare occurrence.

Let us turn now to the consideration of aneurisms of the basilar artery. Twenty-four cases were collected from the literature. Of 21 of these cases in which the sex was noted, 14 occurred in the male sex and 7 in the female sex. The age in 7 ranged between thirty and thirty-seven years, in seven between fifty and sixty-eight years, in six between sixteen and thirty years, and in one case the age was forty-two. According to Lebert[10] most of the cases of cerebral aneurism occur between 40 and 60 years of age, then between 30 and 40, after which between 15 and 20 years.

No symptoms were present in some cases (Grimshaw,[11] Semple,[12] Greenfield[13]), or only headache (Bramwell,[14] Serres,[15] Hodgson,[16] Durand[17]), or only symptoms of short duration (Oppolzer,[18] Bartholow,[19] Eager,[20] Varrentrapp,[21] Bramwell, Durand and Fuller[22]).

In this connection it is interesting to note that in 555 cases of cerebral aneurisms, collected by Beadles, 92 presented no symptoms.

Hemiplegia or paraplegia is rare in cases of basilar aneurisms, having occurred in only 7 of the 24 cases collected. The paralysis was either of long duration (Durand, Watson,[23] Russell,[24] Berger,[25] Beadles, and Van der Byl[26]), or followed a seizure which ended shortly in death (Gordon,[27] Durand, Bramwell, Eager, Oppolzer and Fuller).

Headache, or some subjective disturbances in the head occurred in nine of the twenty-four cases (Gordon, Bartholow, Bradford, Bramwell, Serres, Gull,[28] Hodgson, Durand and Eager).

In 18 of the cases death occurred from rupture of the aneurisms (Gordon, Bartholow, Bradford, Beadles, Grimshaw, Bramwell, Semple, Serres, Van der Byl, Gull, Durand, Blachez,[29] Eager, Varrentrapp, Oppolzer and Fuller).

It should be noted that of the 555 cases of cerebral aneurisms collected by Beadles, 339 ruptured.

The cranial nerves were involved rarely. Of the 24 cases collected by myself, in one there was atrophy of the optic nerves (Watson), and in one the papillæ were swollen.

In two of the cases the facial nerve was palsied (Bartholow, Loomis[30]), and in one case the third and sixth nerves were paralyzed, due to the hemorrhage (Bartholow).

Deafness was noted only in three of the 24 cases collected. It occurred in 6 of the 31 cases collected by Lebert. The deafness is said to be due to obliteration of the artery supplying the middle ear as well as in part to pressure upon the nerve.

The size of the aneurisms of the cases collected varied from that of a pea to that of a pigeon's egg. As has already been said, the size of the aneurisms has little to do with the severity of the symptoms, as a large aneurism may cause no symptoms as my case indicates.

Basilar aneurisms occur usually according to Durand on the superior part of the artery. He quotes Lebert as saying that basilar aneurisms develop principally in front and above or posterior and beneath the artery.

A word as to the frequency of cerebral aneurisms. Gowers classified the cerebral aneurisms as follows in the order of their frequency: Sylvian, basilar, internal carotid, the artery of the corpus callosum, posterior communicating, anterior communicating, vertebral, posterior cerebral and inferior cerebellar.

Krey[32] found in 142 cases of cerebral aneurisms collected from the literature that the site of the aneurism was the basilar, 42 times; middle cerebral, 38 times; the internal carotid, 23 times; the anterior cerebral, 11 times; the posterior communicating, 9 times; the posterior cerebral, 6 times; the middle meningeal, 2 times; the superior cerebellar, 2 times; the inferior cerebellar, 1 time.

In the opinion of Bradford,[33] after the middle cerebral artery the basilar artery is the most frequent seat of cerebral aneurisms. According to Nonne and Luce[34] aneurisms of the carotid and vertebral artery occur most frequently after which the basilar artery is the most frequent seat of aneurisms.

I think it may be concluded that there are no distinguishing features in the symptomatology of cerebral aneurisms. Beadles believed that it was impossible to make a positive diagnosis of cerebral aneurisms, except under the most unusual circumstances. Even when definite pressure symptoms are presented, there is no way of excluding the possibility of a cerebral growth. While Beadles believed that symptoms of cerebral aneurisms are characterized by intermittency occasionally, even this is rare. I believe that the paralyses which occur in cases of cerebral aneurisms which are of long duration are more frequently due to concurrent processes in the nature of foci of softening, or specific lesions such as were seen in the case reported in this paper, than to the pressure of the aneurism itself.

Pressure of tumors at the base of the brain often causes no symptoms, probably due to the fact that the brain tissue becomes displaced rather than compressed.

LITERATURE.

2. Morgagni in Killian. Inaug.-dissert. Wurzburg, 1879.
3. Beadles. Brain, 1907, p. 285.
4. Mitchell. Jour. Ner. and Ment. Dis., 1889, p. 44.
5. Bramwell. Clin. Studies, 1905–1906, No. 4, p. 289.
6. Epron. Contrib. a l'étude d. anevrysmes, etc., These d. Paris, 1890.
7. Bruns. Die Geschwulste d. Nervensyst., Berlin, 1897.
8. Oppenheim. In Beadles.
9. Gowers. Diseases of the Nervous System, 1896.
10. Lebert. Berl. klin. Woch., 1866. p. 209.
11. Grimshaw. Brit. Med. Jour., 1873, p. 213.
12. Semple. Trans. Path. Society, London, 1869, p. 109.
13. Greenfield. Trans. Path. Society, London, 1878, XXIX, p. 91.
14. Bramwell. Clinical Studies, 1907–1908, p. 398.
15. Serres. Arch. gen. d. médecine, 1826, p. 419.
16. Hodgson. Diseases of the Arteries and Brain, 1815, p. 76.
17. Durand. Anevrysmes d. Cerveau, 1868, p. 46.
18. Oppolzer. Wien. med. Woch., 1860, p. 132.
19. Bartholow. Am. Jour. Med. Sciences, 1872, N. S., 6, p. 274.
20. Eager. London Med. Gazette, 1846, vol. III, p. 1384.
21. Varrentrapp. Arch. d. Heilkunde, 1865, Vol. VI.
22. Fuller. Trans. Path. Society, London, Vol. XVI, p. 83.

23. Watson. London Lancet, 1888, Vol. II, p. 718.
24. Russell. Brit. Med. Jour., 1870, p. 87.
25. Berger. Bull. d. l. Soc. Anat., 1869, No. 44, p. 188.
26. Van der Byl. Trans. Path. Society, London, 1858, Vol. VII, p. 122.
27. Gordon. Med. Times, Vol. XIX, p. 381.
28. Gull. Guys Hosp. Reports, 1859, Vol. V, p. 297.
29. Blachez. Bull. d. l. Soc. Anat., 1862, p. 335.
30. Loomis. In Bartholow.
31. Gowers. In Sergent and Grenet-Let Bull. Med., 1908, p. 538.
32. Krey. Inaug. Dissert., Greifswald, 1891.
33. Bradford. London Lancet, 1894, No. 2, p. 913.
34. Nonne and Luce. Handbuch. d. pathol. anat. d. nervensyst., 1904, Vol. I, p. 215.

CEREBELLAR SYMPTOMS AND CEREBELLAR LOCALIZATION

INCLUDING KINEMATOGRAPHIC OBSERVATIONS ON CEREBELLAR PHENOMENA *

CHARLES K. MILLS, M.D.
Professor of Neurology in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital

AND

THEODORE H. WEISENBURG, M.D.
Professor of Neuropathology and Clinical Neurology in the Medico-Chirurgical College of Philadelphia; Neurologist to the Philadelphia General Hospital

PHILADELPHIA

RECENT ADVANCES IN CEREBELLAR SYMPTOMATOLOGY

Our object in this article is to give in condensed form the present status of cerebellar symptomatology and localization, basing the paper on a study of the recent literature of the subject and our personal investigations of a series of patients, both by the ordinary clinical and clinicopathologic methods and by the use of moving pictures. For the most part moving pictures have been used hitherto for the purpose of demonstration in teaching. We believe, however, that they have a value as a method of investigation, which is especially exemplified in the study of cerebellar phenomena. The cerebellum is a strictly motor organ, and many of the symptoms of cerebellar disease, owing to the enlarged luminous image and the repetitive representation of special manifestations, can be studied more fully than by direct examination of the patient. Motor aberrations are strongly impressed and they can be more fully dissected into their elements by the prolonged observation.

To the papers of Rothmann and Babinski, and of Babinski and Tournay, in their contributions to cur-

---

* Read before the Section on Nervous and Mental Diseases at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

rent medical literature and as presented at the Seventeenth International Congress of Medicine held at London in August, 1913, we are much indebted. Among others to whom we owe the most are Ferrier, Bárány, Bolk, Bing, Thomas, and Stewart and Holmes. It is not within our purpose to give an extensive bibliography of the subject. Those especially interested will find it desirable to consult the bibliography appended to the paper of Babinski and Tournay in the *Proceedings* of the Seventeenth International Congress, which contains references to no less than eighty-four titles.

Until the additions of very recent years the symptom-picture of disease of the cerebellum presented in our text-books, treatises and most other writings on the subject had assumed a form so set as to be entitled to the rather abused designation of "classical." This included uncertain station, the patient being usually described as swaying, with legs apart and arms abducted; a gait wabbling or staggering, often described as titubating or like that of a drunken man; forced movements, lateral, forward or backward; ataxic movements of the limbs, these being steadied with the patient recumbent; nystagmus and vertigo usually, and peculiar disturbances of speech sometimes being included.

This symptomatology was one mainly attributable to cerebellar deficit, to the phenomena resulting from destructive lesions. Following Luciani and Weir Mitchell, it has been much the custom with writers on the cerebellum to lay stress on the difference between symptoms of deficit and those of irritation. This differentiation may have been carried too far. In experiments on animals, symptoms of irritation are sometimes shown, but when the lesions experimentally produced include extirpation, the symptoms of deficit usually rapidly replace those of irritation.

In recent years, and especially under the lead of Babinski and Rothmann, some features of the old cerebellar symptomatology have come to be looked on in a different light and some of the symptoms regarded as elementary have been subjected to considerable dissection. The so-called cerebellar ataxia, now regarded as a symptom complex rather than as a pure symptom, has been more clearly differentiated from the ataxia of tabetics. Luciani's astasia, asthenia and atonia are acquiring a new interpretation.

Our studies emphasize the fact that asynergy is the fundamental symptom of cerebellar disease.

Cerebellar tremor and the new symptoms, hypermetry and adiadokocinesis, all now recognized as important cerebellar disorders, when reduced to their symptomatic elements, are seen to be dependent in large part if not entirely on loss or disturbance in the power of performing more or less complex movements together for the accomplishment of a definite end.

Luciani's asthenia, atonia and astasia are to be regarded not so much as special manifestations of asynergy as derivatives or resultants of asynergic effort. Asthenia is due to the exhaustion which results from efforts to perform movements which cannot be properly grouped and directed. Cerebellar atonia, which is very irregular in its expression, is a relaxation dependent on the fact that tonectic stimuli from the cerebral cortex cannot rhythmically combine with unsynergized movements. Primarily, tone is a cerebral, not a cerebellar attribute.

Attention may be fixed on the views just expressed by the following tabular statement:

Fundamental Symptom in Cerebellar Disease:

- Asynergy.

Special Symptomatic Manifestations of Asynergy:

- Hypermetry.
- Adiadokocinesis.
- Tremor.

Symptoms Resulting from Asynergic Efforts:

- Asthenia.
- Atonia.
- Ataxia.

Whether vertigo and nystagmus, which are so often present in disease of the cerebellum, are ever true cerebellar symptoms is a question which has been much discussed. From the time of Flourens' early experiments, physiologists and clinicians have concerned themselves with the relations between labyrinthine or vestibular nerve phenomena, and those dependent on pure cerebellar activity. Comparisons and parallels have been drawn and the extraordinary similarity in some instances and even the identity between the results of experiments on the vestibular nerve apparatus and the cerebellum have been shown by many competent investigators. The decision whether vertigo and nystagmus are cerebellar or vestibular symptoms must be largely determined by a thorough study of each case.

From the position of the cerebellum and its peduncles, the cranial nerves and particularly those from the fifth to the twelfth are frequently implicated in cerebellar affections, the complex resulting being compounded of true cerebellar symptoms and those referable to such of these nerves as may be involved. To this type of case Stewart and Holmes have given the name of "extracerebellar." The well-known and much-studied affections of the cerebellopontile angle belong in this category, although extracerebellar symptoms are not limited to the phenomena of lesions of this angle.

Besides the cranial nerves, the nuclei and the tracts of the neuraxis are not infrequently invaded in cerebellar lesions, and we have still another combination of symptoms, cerebelloneuraxial, which requires close scrutiny in the process of diagnosis.

The following tabulations will assist in fixing attention on the cranioneural and neuraxial phenomena which may be present in association with cerebellar disease:

Extracerebellar Cranial Nerve Symptoms:

Vertigo nystagmus (vestibular nerve).
Deafness, tinnitus (cochlear nerve).
Facial paresis or spasm (seventh nerve).
External strabismus (sixth nerve).
Pain, anesthesia in face, etc. (sensory fifth nerve).
Paralysis of jaw movements (motor fifth nerve).
Pain, anesthesia and paralysis in mouth and pharynx (ninth nerve).
Laryngeal, respiratory and cardiac disturbances (tenth nerve).
Paralysis or spasm of tongue (twelfth nerve).

Extracerebellar Neuraxial Symptoms:

Paresis or paralysis of limbs (pyramidal tract symptoms).
Spasticity of limbs (pyramidal tract symptoms).
Convulsions, unilateral or general (pyramidal tract symptoms).
Changes in reflexes (pyramidal tract symptoms).
  Plantar reflex variable (Babinski response sometimes present).
  Knee-jerk variable.

Sensory tract symptoms: alterations of
  Muscular, tactile, pain and temperature senses.
Disorders of associated ocular movements.
Pupillary, secretory and vasomotor symptoms (sympathetic system).

In this paper, necessarily brief and intended to be elementary, we shall not be able to go into a detailed consideration of special cerebellar symptoms. To a few of these, however, attention will be directed.

The clinician is indebted to Babinski for the fixation of his attention on cerebellar asynergy. Ferrier, however, recognized the fact that synergic control was the particular rôle of the cerebellum. Discussing the mechanism of cerebellar coordination and the relative development of the cerebellum, he says:[1]

> Every form of active muscular exertion necessitates the simultaneous cooperation of an immense assemblage of synergic movements throughout the body to secure steadiness and maintain the general equilibrium; and on the hypothesis that the cerebellum is the center of these unconscious adjustments we would expect the cerebellum to be developed in proportion to the variety and complexity of the motor activities of which the animal is capable. The facts of comparative anatomy and development are entirely in harmony with this hypothesis.

Synergy or synergia is the power or faculty by which movements more or less complex, but functionally definite, are associated in a special act or acts. In a broad sense it is motor association, this association being carried out in movements of different parts of the same limb or in synchronous movements of the trunk and limbs, or of the head and trunk and limbs, etc.

Babinski in one of his papers speaks of asynergy and inertia as the primordial cerebellar symptoms. It seems to us that even inertia can be regarded as what might be termed a negative illustration of asynergy. The inability to move because of cerebellar deficiency is dependent on the fact that the motor adjustments necessary for movements cannot be initiated. Movements are mismeasured, giving dysmetry or hypermetry because the patient with cerebellar disease is unable motorially to associate the different elements of such movements. Adiadokocinesis, as illustrated for example by the impossibility of correctly and rapidly pronating and supinating the hand on the forearm, is due fundamentally to the inability to carry out fine motor associations of an antagonistic kind.

---

[1] Ferrier, David: Functions of the Brain, Ed. 2, 1883, p. 200.

The method usually employed in clinical study or class instruction of demonstrating hypermetry is the well-known finger-to-nose test; for adiadokocinesis, partly flexing the forearm or forearms on the elbow and then rapidly pronating and supinating the hand, the movement being largely one from the wrist. Adiadokocinesis and hypermetry are both demonstrated by the rapid pronation and supination of the hand on the knee. Tremor is exemplified by the finger-to-nose test and by efforts to grasp an object or by writing. Hypermetry is shown by the finger which usually is able to touch the end of the nose overshooting to one side, or in the hand-knee test by the hand not keeping within the normal range of movement. The patient exhibiting adiadokocinesis performs the movements of pronation and supination on the knee or of the semiflexed forearm, hand and separated fingers in a slow, uncertain and awkward manner.

The phases of asynergy described as dysmetry or hypermetry are discoverable, however, in cerebellar cases in the abnormal attitude or movements of the head, in the bending of the trunk, in the abduction, sprawling, overstepping or understepping in walking, in the spreading of the limbs in standing and in much else that is done by the patient, and these dysmetric phenomena are frequently accompanied by tremor and adiadokocinesis, all of which are well defined in kinematographic observations.

KINEMATOGRAPHIC OBSERVATIONS

Our kinematographic investigations indicate the necessity of some revision of views both as regards the methods of describing and the intrinsic characteristics of cerebellar symptoms. Some new points cerebellar symptomatology were elicited. It is to be regretted that we are not able to reproduce in an article the kinematographic representations except by a method so elaborate and so expensive as to make it prohibitive. One must actually see the pictures in order to appreciate their value.

The *gait* of a cerebellar patient in the majority of cases is chiefly trunkal. The trunk may go backward, or forward, forward and to one side, or simply to one side. The inclination may be total or partial. In every attempt on the part of the patient to walk, for example, the trunk from the first leans backward, forward or to one side, it being impossible for him to stand for any length of time in the erect posture. He may be able, however, for a short time to stand erect, or to walk either with the trunk erect or leaning, but even when this is possible he is likely soon to lurch forward or backward or to one side. In this type of case the movement of the trunk is predominant and forms the most impressive part of the picture, the legs seeming to act simply as props for the trunk. The trunk leads, and whatever way it goes, the legs follow. If, for example, the trunk leans forward and to the left, the swing of the left leg is shorter than that of the right, or if the trunk goes forward and to the right, the swing of the right leg is less than that of the left, the legs accommodating themselves to the movements of the trunk.

Our observations on pelvic girdle and shoulder girdle movements in locomotion, both when the patient is standing and when on all fours, are especially instructive. In some of the cases the asynergy is just as marked in the shoulder girdle as in the pelvic girdle. In those cases, however, in which the asynergy is more marked in the pelvic girdle, even if both girdles are affected, the gait is entirely different. Here the legs are held farther apart and the arms are nearly always held partly outstretched and semiflexed at the elbows. If the asynergy is shown alone in the pelvic girdle movements, the trunk assumes a different relation to the legs than in the cases just described. Here the trunk is held rigidly erect and the legs are pushed forward from the pelvis, giving the impression that the trunk goes after the legs. In the extreme pelvic girdle cases, it is impossible for the patient to walk without help, but when assisted, the trunk is very rigid, the legs being thrown out in almost any direction.

The cerebellar gait is often described as drunken or reeling, but from our observations, the gait is not like that of one who is drunk. A drunken man will reel in any direction, but he does this largely because of his mental, that is, of his cerebral reduction. A slightly drunken man simply reels forward or to one side, while one who is very drunk is unable even to stand up and any attempt on his part to do so brings on a sort of crumpling of the whole body. In such attempts the legs give way and the man falls because of this and not because his trunk forced him in a given direction. Besides, there is no attempt on the part of a drunken person to hold his arms outstretched for protection, whereas, the cerebellar patient is always careful as to how he walks, and knowing that he tends to stagger, makes efforts to obviate this and assumes as far as he can protective positions. He not only uses his cerebrum, which is largely out of commission in the case of the drunken man, but he even compensates in some degree for his cerebellar impairment by the forced or overuse of his cerebrum.

As has already been mentioned, when a cerebellar patient walks, the whole trunk may be projected in a given direction or one shoulder or one side may be moved more than the other, as for example, when a patient walks forward and to the left, in which case the left shoulder and left trunk are forward, the right side of the body lagging. The same thing will be true no matter what other position the body assumes, as when the patient changes to a sitting or standing from a recumbent posture. If the case is left-sided, then the left shoulder and trunk move first and more than the right; if right-sided, the right shoulder and trunk are more in the picture. If the case is bilateral or central, that is entirely trunkal, no difference in the movement of either side is detectable.

In the pelvic girdle cases when the patient attempts to get up from a recumbent posture and temporarily assumes in doing this a position on his hands and feet, the sway of the pelvis is very marked and may be chiefly to one side, but is nearly always to both sides. It is noticeable that in this position, the taking of which is one of the best methods of showing the relative degrees of pelvic girdle and shoulder girdle asynergy, if the pelvis only is involved, the trunk will not sway and whatever trunkal movements are present, are compensatory. These cases are much more common than those of the shoulder girdle. The methods thus indicated of demonstrating pelvic girdle and shoulder girdle asynergy have not, we think, been described before.

Turning now to some of our observations regarding *the movements of the limbs,* we are convinced that in testing for hypermetry, for example, it does not matter very much whether the finger goes beyond the nose, for many patients because of being repeatedly asked to do this learn to put their fingers to their noses quite well. The important thing is that if there is an asynergy of the upper limb when the patient attempts to place his finger to his nose, the movement being of wide range as when it starts with the hand on the knee, the whole arm including the shoulder takes part in the movement. The circle described by the hand is greater than it should be and in the majority of cases the movement is quicker than normal, although in some it is the contrary. In some cases when the patient is asked to put his finger to his nose the arm is swung inward; in some cases, outward and in some directly to the nose. The more marked the dysmetry the greater is the tendency for the sweep to be inward, that is, toward the body, and the greater is the involvement of the shoulder.

In testing hypermetry in the legs, if the patient is in a recumbent position and asked to put his heels to his knees or his heel to any point indicated, there is first an overflexion of the thigh on the abdomen and the heel nearly always overshoots the desired point. The nearer the pelvis is the point to be touched, the greater the sway of the leg, but the heel is usually placed on the desired point.

An equally good way to test hypermetry is by asking the patient to put the heel to the buttock. In this the movement is nearly always quick, the thighs are much more flexed on the abdomen than they should be and in the height of the excursion, with the heel approximating the buttock, there is always a to-and-fro sway of the limb, the degree of this depending on the severity of the symptom.

In testing for adiadokocinesis the attempts at alternate pronation and supination of the forearm and hand quickly show the degree of asynergy of the limbs. Adiadokocinesis may be tested in the lower limbs as well as the upper by alternate rotations or adductions and abductions. The defect is readily observed. The excessive movement may be either inward, outward, upward or downward in the upper limb and inward or outward in the lower limb. This is best brought out if only one side is involved, for then it is possible to compare the movements with those of the limbs of the other side. In some cases the movements are slow, but the important thing to look for is the overmovement.

In a case recently placed on record by one of us there was a *loss of emotional expression* on the right side of the face although the patient could use this side for every other purpose. The loss of emotional expression, while evident to ordinary observation, was emphasized by the kinematograph. In other cases, as for example, in paralysis agitans, disseminated sclerosis and focal lesions of the pons, we have been able to study changes in emotional expression more thoroughly than by the ordinary clinical method.

Not only can unilateral loss of emotional expression be exhibited, but a loss of all or almost all expression. One of our patients who could not be made to laugh or cry talked in a slow, monotonous, stupid manner with drooling of saliva. The moving pictures show not only the loss of emotional expression, but also the manner of speech. It is interesting that in this patient the movements of the limbs and body are of the same character as those of speech.

We believe in a true *cerebellar speech,* one type of which is shown in the patient described above. In another case which was for a long time in the nervous wards of the Philadelphia General Hospital the history was such as to indicate that the patient had had a hemorrhage into the anterosuperior portion of his cerebellum. He had most exquisite asynergy in his tongue, facial and laryngeal movements. His speech might be described as jerky and crackling. In this patient and in one other whom we recall, not only was the speech affected in the manner described, but mastication and deglutition were almost impossible because of the asynergy of the involved musculature.

In most of our cases examined kinematographically there were no asynergic *movements of the head and neck.* The head in these cases usually maintains the same plane as the trunk. In one case, however, shown in the series of pictures, the head is carried distinctly to one side. While the cases seemed to be relatively limited the fact that we have had a few speaks for the localization of movements of articulation, phonation, mastication and deglutition.

So far as our kinematographic observations are concerned, we showed, in one case, previously mentioned, of cerebellar agenesis, what we regard as true *asynergia of the eyeballs*. The eyes are never at rest. They move upward, downward, to either side, and in a rotary direction and combined, with the nodding of the head, give a furtive appearance, as if the patient were constantly dodging something. No two successive movements of the eyeballs are alike and it is with difficulty that the movements can be followed with the naked eye, although with the kinematograph this becomes easy.

In one of our patients vertical nystagmus was always present, and this occurred no matter in what direction the patient attempted to look. We believe because of the symptomatology that this patient had a lesion of the upper cervical cord and inferior cerebellar peduncles. We regard the nystagmus as due to involvement of the vestibulocerebellar apparatus, probably of the parts connecting the vestibular nuclei with the deep nuclei of the cerebellum.

In one of our patients in whom an exposure of the cerebellum was made, the cerebellum was incised to about the depth of half an inch at about the junction of the left superior semilunar lobe with the vermis. Directly after the operation and ever since, marked lateral jerkings of both eyeballs have been constant, but these have been more marked on the left. If the patient looks to the right the nystagmic movements are not so forcible, but on looking to the left they are strikingly exhibited. It is not that the left eyeball moves without the right, but that the excursion in the left eye is more marked than in the right and gives one the impression that the left eyeball alone is moving. It will be remembered that the incision was on the left. As these movements were not present before the operation it is apparent that they were the direct result of the incision into the cerebellum.

Babinski has described catalepsy as a new cerebellar symptom. In demonstrating this the patient is placed on his back, the thighs semiflexed on the abdomen, the legs on the thighs. After a few oscillations, according to Babinski, the legs become stationary and maintain their position for a longer than normal period. These phenomena in one patient were shown by Babinski by means of a moving picture at a meeting of the International Congress, 1913.

We found that most of our patients were too asynergic and asthenic to maintain their legs in the above-described position for more than a few moments. In other patients in whom the asynergy was not so marked, and particularly in the unilateral cases, we tested both the upper and lower limbs at the same time. The upper limbs in this test were first placed in a vertically extended position. The limbs on the affected side slowly drooped and reached the level of the body sooner than those of the unaffected side. In not a single patient did we find true catalepsy and we therefore do not look on it as a manifestation of pure cerebellar disease. In one of our patients, a very powerful man, one of our chief illustrations in the series of moving pictures, the man placed on his back was able to maintain his legs and arms in a set position—that of semiflexion of the lower limbs and vertical extension of the upper—but in this case there was no preliminary swaying and we regard the exhibition as pseudocataleptic and simply due to the great strength of the individual. The case was one in which the asynergic phenomena were expressed bilaterally, although the abnormal movements were more marked on the left and especially in the pelvic girdle.

With Dr. Isaac H. Jones we have made kinematographic observations on both normal patients and those suffering from cerebellar disease with the Bárány and other tests.

CEREBELLAR LOCALIZATION—FOCAL SYMPTOMATOLOGY

In studying the focal symptomatology of cerebellar disease, hypermetry, adiadokocinesis and tremor at times indicate not only the presence of cerebellar asynergy, but give the clue to the part of the cerebellum most involved, as when these phenomena are most markedly exhibited in a special part of the body —the trunk, the upper or lower extremity, the head, face or eyes. These facts of localization are, we believe, emphasized by kinematographic investigations, as we have just tried to show.

The clinician soon finds that the focal symptomatology of cerebellar disease is not so easily comprehended as cerebral focal symptomatology. He recalls, however, from his experience, observations which have a bearing on this question. He has had cerebellar cases with marked unilaterality in their manifestations, indicating that one-half of this organ exerts its influence with marked predominance on one side of the body. In other cases, not on the whole numerous, ocular movements have been disordered in a way not explicable by limited lesions of parts of the brain other than the cerebellum. In other instances, also not numerous, the speech of the patient is affected in a manner not to be explained merely by bulbar or cerebral involvement, while in many instances both ocular movements and speech disturbances are wanting. He has noted that in different cerebellar patients the disordered movements of the trunk differ from each other in ways which can be definitely discriminated. Shoulder girdle movements in one instance, and pelvic girdle movements in another, are more affected. The changes in emotional expression sometimes observed differ in different cases. The head takes a special attitude in a few cases; in many others this not occurring. Extraordinary combinations of abnormalities of movement may be discoverable on casual observation or elaborate investigation.

The cerebellum as a whole and all of its parts are concerned with the function of synergia. When, however, a particular part or parts of the body are the seat of abnormal motor phenomena of cerebellar origin, such manifestations are focal and more or less capable of localization. If these focal symptoms were arranged in tabular form, we would have a list something like the following:

Focal Symptoms Indicated by Localized Asynergic Movements:

Eye movements.
Attitude and movements of head.
Movements of jaw, face, tongue, larynx, etc.
  (Disturbances of speech, emotional expression, etc.).
Position and movements of trunk.
Special movements of upper extremity.
  (Disturbances in fine movements including writing).
Movements of trunk and upper extremity (shoulder-girdle movements).
Special movements of lower extremity.
Movements of trunk and lower extremity (pelvic-girdle movements).

CEREBELLAR LOCALIZATION—COMPARATIVE ANATOMY AND EXPERIMENTATION

Some of the results of embryologic and morphologic investigation of mammalian cerebella indicate the development and elaboration of function in this organ. Among those who have contributed work of great value in this direction we find some of the most distinguished names in the field of neuro-anatomy: Kölliker, Schwalbe, Elliot Smith, Bolk, Van Rynberk, Charnock Bradley and others. To the results of the investigations of some of these, Rothmann has appealed in corroboration of his own experimental and pathologic findings.

Studying the mammalian cerebellum from the lowest types to man, certain facts of paramount interest regarding the development of fissures, folia, lobules and lobes attract attention. The methods in which fissures appear, extend, recede and disappear and the horizontal and sagittal expansion of cerebellar substance go hand in hand, as in the development of the cerebrum with additions and intensifications of function.

In the lower mammals like the squirrel, the ox, the dog and the monkey, the cerebellum has a less marked subdivision into a middle lobe and lateral lobes than in man. Such subdivision appears and becomes more definite as the evolutionary series approaches man. Glancing, for example, at the dorsal aspect of a series of the cerebella of apes or their semidiagrammatic representation as given by Bolk, one deep somewhat anterior fissure usually called the *sulcus primarius* is uniformly observed traversing the entire width of the cerebellum, while above and below this are distinct fissures shallower and of varying lateral extent, only one, much more posterior than the primary fissure mentioned above, crossing the full width of the cerebellum. In the cerebella of these animals a vermis of moderate extent is distinctly to be seen in the posterior region of the cerebellum. In *Lemur albifrons* the deep transverse fissure is seen with shallower fissures anterior and posterior to it. In its posterior median portion is a well-defined middle lobe, to the right and left of which are lobes traversed by a short fissure with radiations. This intercrural fissure separates two crura or limbs of a cerebellar lobe, the *crus primum* and *crus secundum*. Many of the references to the experiments on lower animals, as on dogs and monkeys, are to the effects produced by extirpation or irritation of these parts.

Bolk, Elliot Smith and Bradley have made schema of the mammalian cerebellum. Von Bechterew adapted the schema of Bolk so as to give a general view of the functions of the cerebellum (Fig. 1).

It is in the parts homologous in the human brain with the *crus primum* and its correlated vermis that the highly evolved synergic movements of the upper extremity and trunk appear to be represented, and in the *crus secundum* and its correlated median and paramedian regions the highly developed synergic movements of the lower extremity and trunk, those especially concerned with the maintenance of the erect position of man and his ability to perform complex synergic movements in this position.

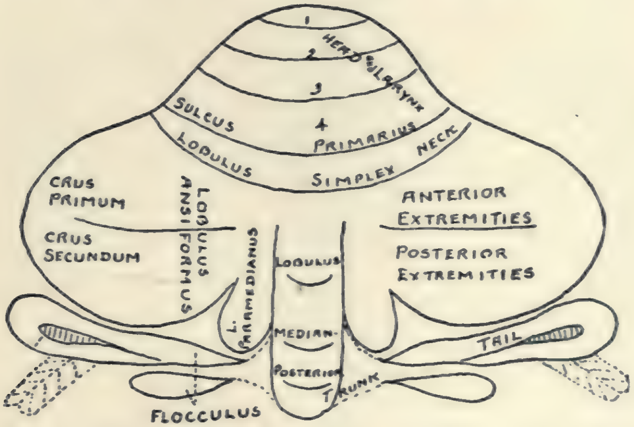


Fig. 1.—Bolk's schema of the mammalian cerebellum; on the left half the new anatomic names; on the right half the functions of special areas (Von Bechterew).

In the anterior portion of the human cerebellum as in lower mammalia, movements more or less common to both them and to man—movements of the eyes, head, jaws, face and larynx—are represented, although this representation in man is of higher synergies than in lower animals. This anterior lobe is to be regarded more as a whole—as transverse or azygos—without so much reference to a separation into middle and lateral parts as has hitherto been considered essential, although the superior vermis and its wings may have subdivisions of their general representation.

The demand for a complex representation of movements which are complex and elaborate has led to the tremendous development in man of that subdivision of the cerebellum which is posterior to the primary fissure.

The localization of cerebellar function and the abnormal phenomena produced by limited cerebellar lesions have been chiefly investigated by extirpation experiments and electrical irritation. The extirpation experiments have been of varying character, sometimes decapsulation of a whole lateral lobe or of this and the vermis or of the vermis alone; and sometimes by extirpation of small areas in definite parts of the cerebellum as in the anterior lobe, in the *crus primum, crus secundum,* the posteriomedian lobe or inferior vermis, or the paramedian region.

The electrical-excitation experiments have been performed on carefully selected spots and areas of the cerebellar cortex and some observers have endeavored to isolate their irritation experiments to the deep nuclei of the cerebellum. We are in accord with Rothmann in our belief that the cerebellar cortex is excitable to the electric current, a fact which was long ago demonstrated by Ferrier and which we have confirmed in one case on a human subject. A stronger or longer-continued current seems to be required to bring about movements in the case of the cerebellar cortex than in that of the cerebral cortex, but such excitation is possible and can be more or less circumscribed.

In another communication, reviewing the neurologic contributions to the Seventeenth International Congress, one of us[2] has summarized the findings indicating special localizations by recent cerebellar experimentalists (Rothman, Prus, Adamkiewicz, Pagano, Bolk, Van Rynberk, Luna, Katzenstein and Rothmann, Bárány, and others). It is not always easy or perhaps possible to homologize the parts isolated in experiments on lower animals with divisions and subdivisions of the human cerebellum, and clinical contributions supported by necropsies have not yet been sufficiently numerous and exact to furnish absolutely dependable corroborations of the work in experimental physiology. Based on our studies of the literature of cerebellar embryology, comparative anatomy, experimentation and clinicopathology and on the results of our direct and kinematographic clinical observations, these studies in a few instances being reinforced by operations and necropsies, we have attempted a tentative schema of localization in the human cerebellar cortex (Figs. 2 and 3). It must be understood that this is regarded by us not only as tentative, but in some particulars as merely suggestive. One cannot with the same accuracy and certainty as in the case of the cerebral cortex indicate this or that region of the cerebellar cortex as exclusively concerned with the movements of the eyes, head, face, jaws, trunk, extremities or special parts of the limbs. The important fact that the cerebellum as a whole and in subdivision is concerned with synergy indicates that movements in which various parts of the body are at the same time concerned cannot be fully represented by a center or area for movements of one of these parts. The eyes may move to the right or left, or up or down without any association with other movements, but on the other hand, the eyes may move synergically with the head or with the head and trunk or with the head and trunk and limbs. The trunk may be bent forward or backward or twisted to the right or left with little or no association with movements of the extremities,

---

[2] Mills, C. K.: Neurology at the Recent (Seventeenth) International Congress of Medicine, New York Med. Jour., 1914, xlix, No. 5.

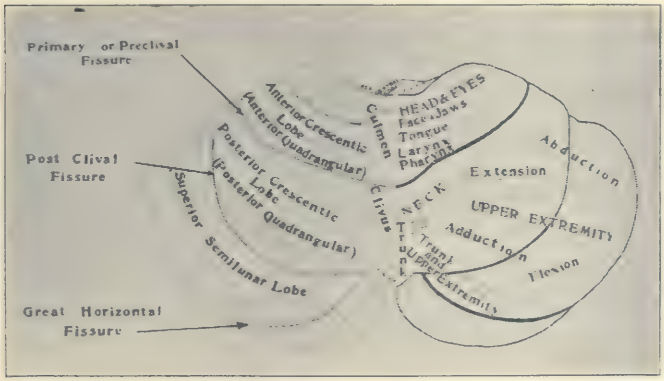


Fig. 2.—Cerebellar localization; zones and centers of the superior surface of the cerebellum.

but such comparatively simple trunkal movements are not the rule in bodily actions. The rôle played by the cerebellum has its best exemplifications in such acts as advancing and retiring in the erect position or in the performance of complex movements while standing, as in fencing, boxing or public speaking; or in the driving and regulating while sitting of the movements of a rapidly running automobile; or in any highly trained movements such as are exhibited in a series ascending in complexity and rapidity of association from a game of tennis or baseball to the handling of an aeroplane. These wonderful representations of instantaneous synergic activity must be represented in the cerebellum by complexly associated neural mechanisms. In other words, cerebellar localization is more compound in its cortical representation than is cerebral localization. A composite, however, has always within it factors or elements which can to a less or greater extent be separated from the entire mass.

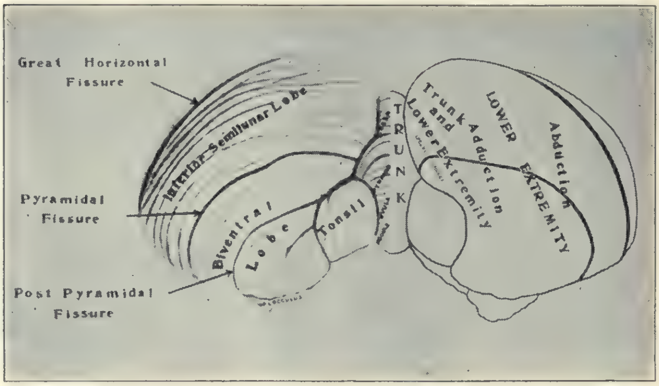


Fig. 3.—Cerebellar localization; zones and centers of the inferior surface of the cerebellum.

In the human cerebellum we must be able to recognize, in the first place, centers for synergically simple movements and in the second place, centers and zones for more and more complex synergic movements. Each simple synergic movements has, however, its special cortical representation.

To illustrate the principles just expounded let us refer to some of the results of experimentation and the difficulty of applying these in ordinary clinical work, at the same time showing that this application is possible. Bolk found a center for bilateral movements of the anterior limbs in the *crus primum* and a similar center for the posterior extremities in the *crus secundum*. A destructive lesion of one of the homologous parts in the human cerebellum would not give us the same simple clinical picture as the lesion of deficit in the dog, this being due, at least in part, to the fact that the dog ordinarily uses all four limbs in station and progression while man step by step with the development of his cerebellum has learned to stand erect and walk and guide his movements chiefly by the motor adjustments between his lower limbs and trunk, the upper limbs acting as auxiliaries in such performances. The aberrant movement resulting from localized deficit or irritation in the case of a man would be more complex, a combination, for instance, of an abnormal shoulder girdle or pelvic girdle movement with specialized dysmetric movements of parts of the limbs. The fact that the representation in certain parts of each hemisphere may be homolateral, contralateral or bilateral makes it more difficult to draw an inference or reach a conclusion as to the localization of function and therefore, of course, as to the results of focal lesions.

How then shall we apply the known facts of cerebellar localization in neurologic diagnosis? Simply by bearing in mind that when the trunkal movements and those movements of the limbs which must act with them to preserve static and dynamic equilibrium are affected, the vermis must be involved in whole or in part. The associated involvement of one lateral lobe in the vermal lesion will be largely determined by unilateral dysmetric phenomena, the extent of the anteroposterior involvement of the inferior vermis by the preponderance of asynergy in either the lower or the upper limbs. In our kinematographic studies the presence of abnormal pelvic girdle movements in one case and of abnormal shoulder girdle movements in another, these symptoms being sometimes more marked on one side than the other, seems to us to give the key to the localization of the lesions both as regards their anteroposterior and lateral positions.

The cerebral representation is of differentiated movements voluntarily performed. The cerebral centers are immediately under the control of the will. They have in them no automatic factor. The higher reflex arc of which each of these centers is a part, has been handed over to the will. Cerebellar representation is more automatic in proportion to the greater complexity of motor adjustments which it represents. The synergic movements controlled by cerebellar centers are not due to volitional mechanisms, although an entire cerebellar mechanism may be called into action by the will. One may turn eyes and head and trunk to the right or to the left or may move forward or backward in any direction, may dance or fence or box or write or perform on a musical instrument because he wills to do this, but in the act of so doing the movements concerned are largely although not exclusively controlled by the cerebellum. The immediate control of synergic movements of whatever sort belongs to the cerebellum. The interrelation between the cerebrum and the cerebellum is, however, close and it is because of this that the cerebrum can so largely compensate for cerebellar loss.

1909 Chestnut Street.—2030 Chestnut Street.

Reprinted from The Journal of the American Medical Association
Nov. 21, 1914, Vol. LXIII, pp. 1813-1818

Copyright, 1914
American Medical Association, 535 N. Dearborn St., Chicago

LE NÉVRAXE

LIVRE JUBILAIRE

DÉDIÉ A

M. A. VAN GEHUCHTEN

Professeur à l'Université de Louvain

A L'OCCASION DU

25me Anniversaire de son Professorat

William G. SPILLER, M. D. (Philadelphie)

BILATERAL OCULOMOTORIUS PALSY
FROM SOFTENING IN EACH OCULOMOTORIUS NUCLEUS

Extrait de la Revue « *Le Névraxe* », vol. XIV, 1913

LOUVAIN
A. UYSTPRUYST-DIEUDONNÉ
10, rue de la Monnaie, 10
1913

BILATERAL OCULOMOTORIUS PALSY FROM SOFTENING IN EACH OCULOMOTORIUS NUCLEUS

Bilateral oculomotorius palsy developing in an apopletic attack, syphilitic in origin, implicating every muscle supplied by the oculomotorius in each eye, resulting from bilateral softening as two separate lesions confined to the nuclei of these nerves; without implication of other cranial nerves except the opticus, and without other symptoms except preceding mental disturbance, convulsions and some change in the patellar reflexes, and succeeding dorsal extension of each big toe from plantar irritation, have made the case reported in this paper worthy of attention.

Officer, a male, aged 52 years, was admitted to the Philadelphia General Hospital March 1, 1904, to the service of Dr. Pearce. He later came into Dr. Mills' service and finally into mine. At the time of admittance the following notes were made:

He had had a fall striking the back of his head nine years previously. As a result of this fall he was unconscious about a week. About two or three months later he had a convulsion, in which he became unconscious and bit his tongue. The attacks have been several in a week, and two or three weeks have passed without any attacks. He was trephined one year after the fall without benefit.

He was a well-built man. The tongue was protruded straight, the station and gait were good with eyes open or closed. The pupils were contracted equally, were regular in outline, reacted in accommodation but not to light. *There were no palsies of any ocular muscles*. The face was not affected. None of the muscles of the body were atrophied. The grip of each hand was good. The patellar reflex was not obtained on the left side, even with reinforcement, but was obtained on the right side, although the leg moved through a smaller arc than usual. Ankle clonus was not obtained on either side. The mentality was slow.

November 11th, 1904. The man had a convulsion about two P. M. on this day and fell striking his head a severe blow over the left eye, this caused a wound requiring two stitches. He could talk and move all parts of his body after the fall, and was able to walk. He showed no bad results from the fall on the following day, and it is doubtful whether it had anything to do with his later condition.

November 18th. He had been acting rather strangely a part of yesterday, rubbing his hands on the carpet and then on his shoes, and doing other peculiar things, though he answered questions rationally. About 7.30 on the morning of this date (18th) he became rigid, had stertorous respiration, puffing cheeks, incoordinate and purposeless movements of hands and upper limbs, and cried out loudly at the beginning of the attack.

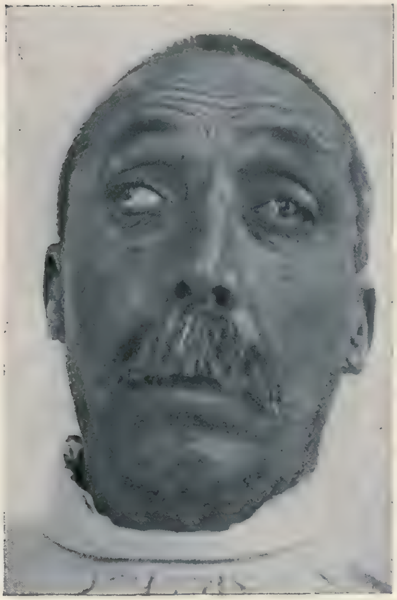


Fig. 1.

Photograph of patient looking directly forward, showing wrinkling of forehead in attempt to overcome ptosis.
Photograph by Dr. Ralph Pemberton.

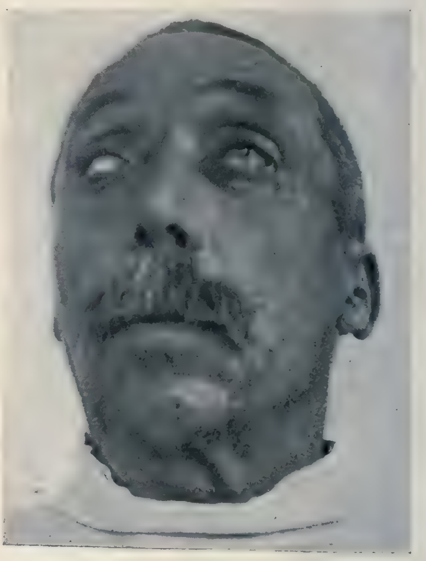


Fig. 2.

Attempt to look upward.
Photograph by Dr. Ralph Pemberton.

When he was examined at 8,15 he was profoundly unconscious, and very rigid in all the extremities. He was catheterized, and the urine showed no trace of albumin. By 3 P. M. he could be aroused by being shaken and spoken to loudly, but he relapsed into sleep again after short monosyllabic answers. On November 20 he was more rational, and the ocular palsies were detected on this date. He had bilateral ptosis with marked compensatory action of the frontalis muscle. He was completely unable to elevate the upper eyelids, and efforts to look upward seemed to increase ptosis. He was unable to converge. Associated movements to extreme right or left were lost. He was unable to move the eyes upward, although the movement downward seemed to be retained, particularly in the right eye. The action of the superior oblique was difficult to determine. The pupils were greatly contracted, and the left was somewhat larger than the right. The movements of the muscles supplied by the facialis and trifacialis were good.

On November 22nd, he had mild delirium, but answered questions. He thought there was a car upside down in the ward, but after a few minutes he recognized his mistake. Dr. SHUMWAY made an examination of the eyes on this date. There was ptosis of both upper lids. The man was unable to raise the upper lids except by the action of the frontalis muscle. Movement of both eyes upward was entirely lost. Convergence movement was lost. Outward movement was preserved although it was somewhat deficient. There was entire failure of motion inward beyond the median line. The movement downward was fairly preserved, and the eyes converged slightly in attempt to look downward, but diverged immediately. The action of the superior oblique was apparently preserved in the right eye, but was very poor in the left eye. The pupils were 2 1/2 mm. There was no response to light or in accommodation. The eyegrounds appeared normal, and there was no evidence of optic neuritis. All who had been in attendance on the patient were convinced that there had been no ptosis previous to the attack on November 18th. It is recorded that on November 28th the voluntary movements in the upper and lower limbs were normal.

A note made December 11th states that the man got out of bed occasionally and walked with comparative ease. He fabricated much. Said he had been out nearly every day, either to market or some other place. When he fixed with the left eye the right deviated externally.

On January 18th 1905 it is recorded that he was considerably clearer mentally, and had been so five or six days. He knew what month it was, recalled past events and realized that he had had no knowledge of what had occurred. He died March 18th, 1905.

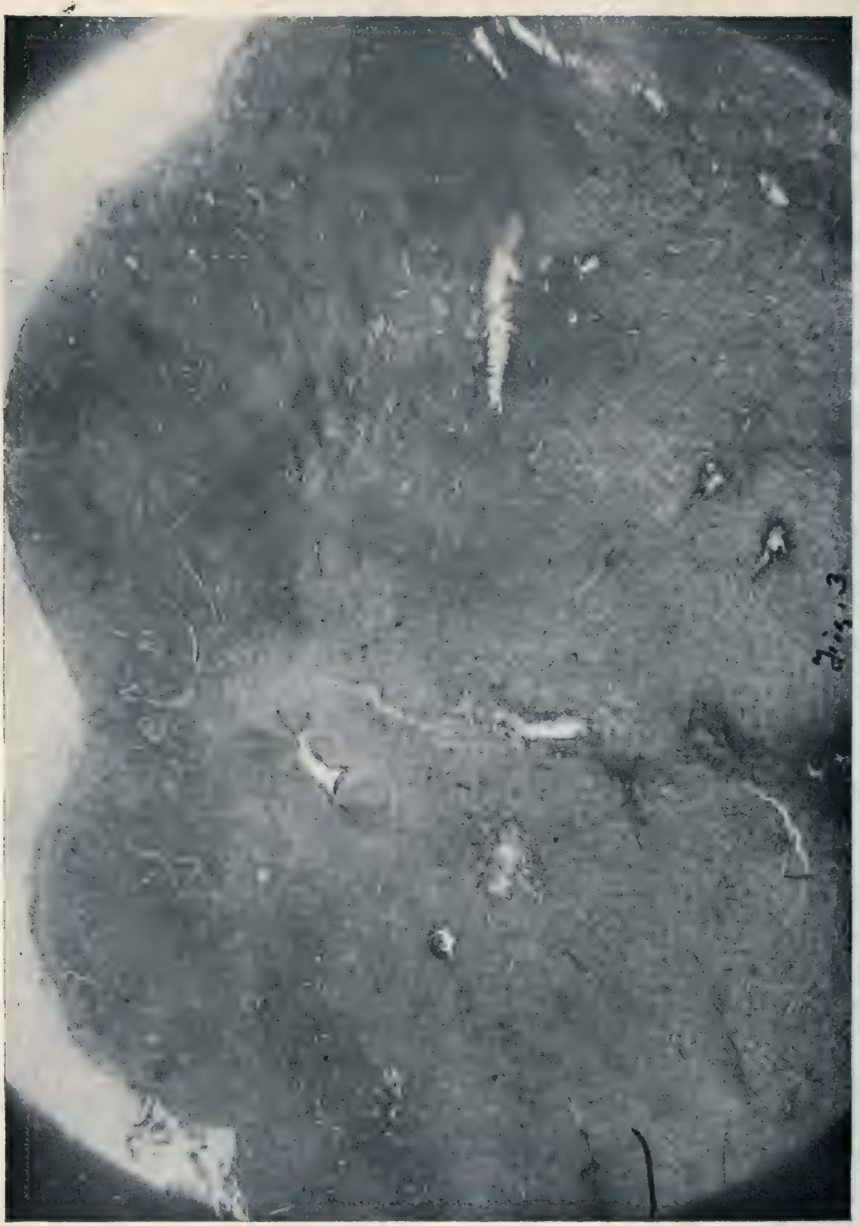


Fig. 3.
Microscopical section under low power showing softening in each oculomotorius nucleus.
Photograph by Dr. A. J. Smith.

The pathological diagnosis was: Chronic pericarditis, chronic pleurisy, congestion and edema of lungs, chronic nephritis.

Sections of the lumbar, thoracic and cervical regions of the spinal cord present a slight round cell infiltration of the pia and about the pial vessels. The pial vessels in places are distinctly thickened, and the intima is proliferated. There is scarcely any round cell infiltration within the cord. The cells of the anterior horns at all levels are well preserved, many show much pigmentation. The posterior columns are slightly degenerated. The pia of the base of the brain shows moderate amount of round cell infiltration.

An area of softening containing fatty granular cells is found in each oculomotorius nucleus in the portion nearest the pons, and is confined to the nucleus. The nerve cells near these areas are greatly degenerated. Small areas infiltrated with red blood corpuscles and hemosiderin varying considerably in size are found about the aqueduct, one larger than the others is found at the periphery lateral to the oculomotorius nucleus, and within this area is a much distended vessel. There is little or no perivascular cellular infiltration at the level of the softenings, such as is common in syphilitic inflammation. The lesions evidently are vascular occlusions. About one to two mm. ventral to the lower part of the posterior longitudinal body is a distended and thrombotic vessel, the wall of which is considerably degenerated, but portions of the elastica are easily recognized. The occlusion of this vessel probably is the cause of the softenings in the oculomotorius nuclei. Near this vessel and a little lateral to it is a smaller occluded vessel. The oculomotorius nuclei are better preserved in their upper portions.

The trunks of the two oculomotorius nerves show much sclerotic tissue, presenting a hyaloid change, this is more pronounced in the left nerve. Many fibers within the nerves are degenerated.

A section of the basilar artery shows much poliferation of the intima on one side.

Both abducens nerves and the right trochlearis are in good candition. Both opticus nerves are greatly degenerated, especially the right.

A large gumma was found between the frontal lobes invading the median surface of each frontal lobe. Many of the microscopical sections were made by Dr. E. Rawlings.

The ophthalmoplegia clearly was caused by thrombotic closure of the small arteries supplying the portion of the oculo-

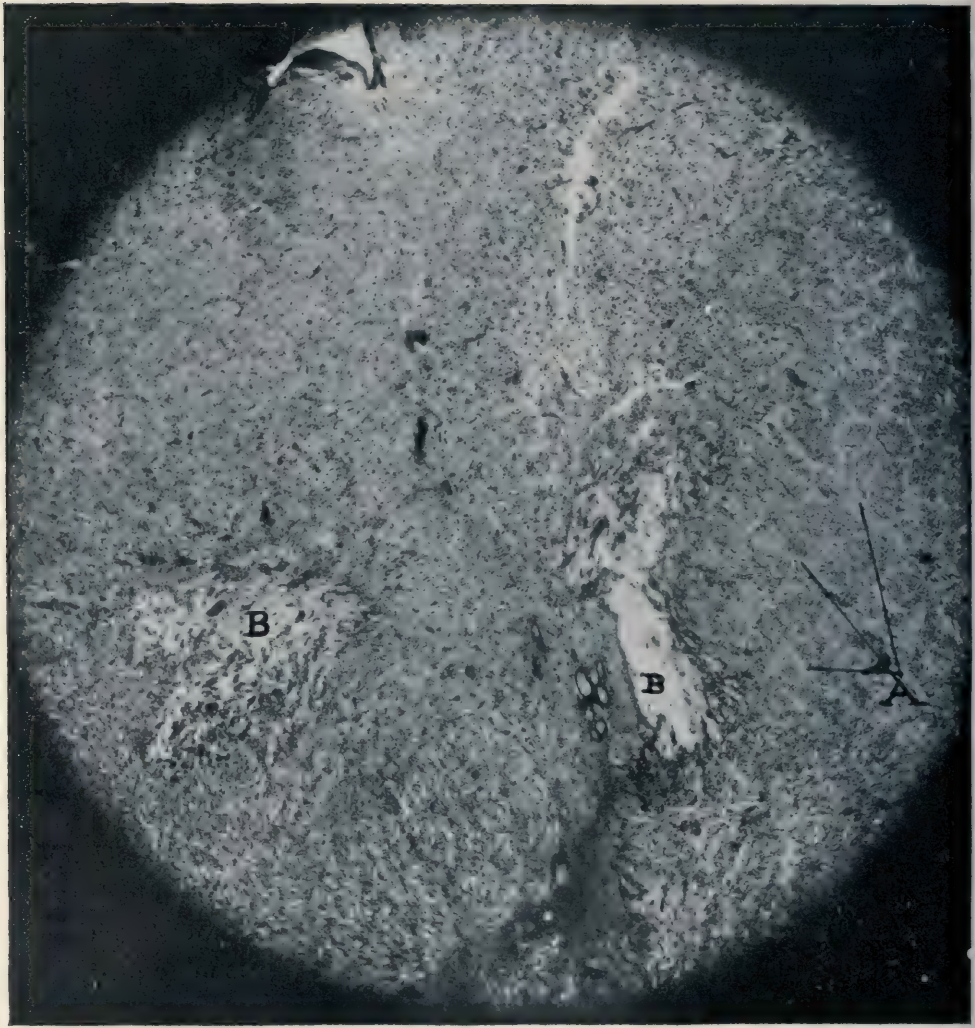


Fig. 4.
The same under greater magnification. A. degenerated oculomotorius fibers.
BB. aera of softening in each oculomotorius nucleus.
Photograph by Dr. A. J. Smith.

motorius nuclei nearer to the pons. The various groupings of symptoms caused by occlusion of certain vessels of the brain have interested me greatly for several years. In June 1908 I reported the findings in two cases of occlusion of the posterior inferior cerebellar artery. At that time with the exception of the work of HUN and VAN GIESON, and H. M. THOMAS, the subject had received no attention from American or English writers, although it had been made familiar by the writings of a few German authors. In December of the same year I described a new symptom-complex caused by a lesion of the uppermost portion of the anterior spinal and adjoining portion of the vertebral arteries. In this complex we should expect to find paralysis of the muscles of all the limbs, trunk and neck, whereas the reflexes necessary to life would be preserved. The face would not be affected. The tongue might escape. Deep sensation, i. e. the so-called muscular sense (the senses of passive movement and position) should be affected. The symptoms are explained by lesions in the regions supplied by the vessels in question, i. e. the anterior and middle portions of the medulla oblongata. A clinical case belonging to this type was reported in this paper. I had hoped that similar cases might be reported by others, but the subject seems to have attracted little or no attention.

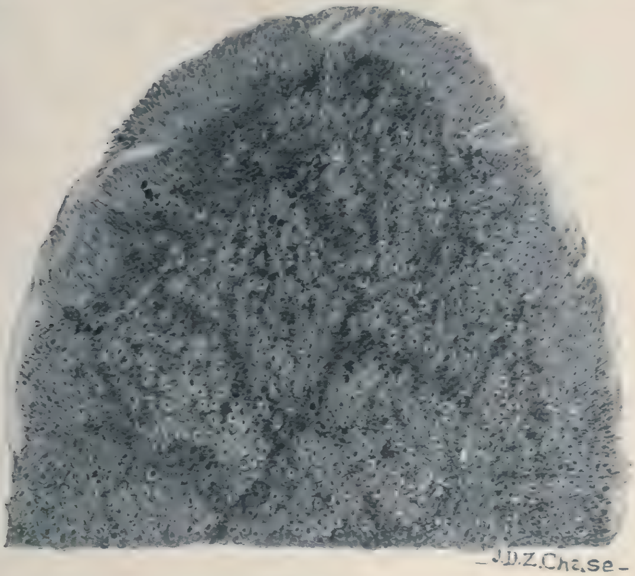


Fig. 5.

Drawing of a section from the left oculomotorius nerve near the cerebral peduncles, showing areas of sclerosis.

In 1909 I reported a case with necropsy illustrating the symptom-complex of occlusion of the cervical anterior median spinal artery (1). I could find no similar case in literature. This case in brief was as follows : A man in good health suddenly experienced pain between the shoulders and felt numb and weak in the upper limbs. Ten or fifteen minutes later he felt numb and weak in the lower limbs. Three or four weeks after the onset he had recovered largely the use of the lower limbs, but these limbs were somewhat spastic. He had normal sensation of touch everywhere. The patellar reflexes were exaggerated and BABINSKI'S reflex was with upward movement of the big toe on the right side. He had much impairment of temperature and pain sensations in the upper part of the thighs, over the trunk as high as the first or second rib, and in both upper limbs. He had incontinence of bladder and rectum. He was almost completely paralyzed in the upper limbs and these limbs were much wasted.

Syphilis seems to be the cause of most of the occlusions of small vessels.

Recently I have reported with Dr. J. W. MCCONNELL (2) a case of bilateral occlusion of the vessels supplying the inner segment of each lenticular nucleus and resulting from carbon monoxide poisoning. Similar cases are known in the literature. The most striking feature of this symptom-complex is great spasticity with a pseudo-paralysis of the limbs. The patient seems paralyzed in all the limbs and yet at times voluntary motion is seen.

In the present paper I am able to show that isolated occlusion of certain vessels to the oculomotorius nucleus may occur, producing symmetrical softening and paralysis of all branches of this nerve, although the paralysis may not be complete in all muscles innervated by the nerve.

Dr. Charles K. MILLS has recently (3) reported a case of occlusion of the superior cerebellar artery.

It is interesting to study the statements of some recent writers who have given special attention to syphilis of the nervous system regarding the frequency of oculomotorius palsy.

---

(1) SPILLER : Journal of Nervous and Mental Disease, 1908 and 1909.

(2) MCCONNELL and SPILLER : Journal of the American Medical Association, 1912.

(3) MILLS : Journal of Nervous and Mental Disease, February 1912.

Mott believes (1) that complete oculomotorius paralysis without any other functional disturbance of the cranial nerves, and only with general cerebral phenomena, is extremely rare. He has seen two cases, both clinical, of brain syphilis in which both oculomotorius nerves were completely paralyzed.

Nonne (2) in his monograph on syphilis of the nervous system gives references to the frequency of oculomotorius palsy. Uhthoff in 167 necropsies of cerebral syphilis (17 of his own and 150 in the literature) found the oculomotorius affected 66 times, the abducens 29 times, and the trochlearis only 6 times. In 259 cases of cerebral syphilis, including those with and those without necropsy, he found the oculomotorius affected 96 times, and concluded that bilateral, isolated, total or partial oculomotorius palsy is more common in syphilis than in any other disease. Alexander found in 146 cases of syphilitic paralysis of ocular muscles the oculomotorius affected in 65 per cent., the abducens in 33.5 per cent., and the trochlearis in 1.5 per cent. Knies found that the oculomotorius was affected in three-fourths of the cases of syphilitic ocular palsy. Uhthoff thought the bilateral implication of the oculomotorius was almost as common as the unilateral, for he found bilateral affection 6 times in his 10 cases, and 22 times in the 56 cases he collected.

Complete paralysis of the oculomotorius, i. e. paralysis of all the external and internal branches is much less common than partial paralysis. Alexander found this nerve completely paralyzed 19 times and partially paralyzed 145 times. Cases of complete oculomotorius paralysis in syphilis have been reported by Alexander, Uhthoff, Oppenheim, Buttersack and many others.

The most frequent lesion producing syphilitic palsy of the oculomotorius is basal meningitis. Gummata have been found in the nuclei of the nerve and its roots, softening from vascular lesions have been described by Alexander, Bristowe, Hughlings Jackson, Leyden and others, but these were not cases of softening confined to the nerve. That syphilitic encephalitis without specific syphilitic products, by which term persumably is meant gumma, may exist is shown by the report of a case by OPPENHEIM and of one by WILBRAND and SAENGER. In the first case non-purulent encephalitis was found, in the second case inflammation about the aqueduct of SYLVIUS was found; the vessels were enlarged and infiltrated with round cells.

---

(1) Mott : *A System of Syphilis*. Power and Murphy, vol. 4, p. 72.

(2) Nonne : *Syphilis und Nervensystem*, second edition, p. 206.

WILBRAND and SAENGER (1) say that primary degeneration of the nuclei of the nerves to the ocular muscles is demonstrated by a case of OPPENHEIM. HUTCHINSON first drew attention to this result of syphilis, and GOWERS made the microscopical study in his case, but the relation of the primary degeneration to syphilis was uncertain and the case appears more as one of tabes. OPPENHEIM'S case WILBRAND and SAENGER regard as the only case they could find in which primary nuclear degeneration occurred during the period of constitutional syphilis. His patient presented many symptoms of nervous syphilis in addition to ocular palsies. Many nerve cells were degenerated in the abducens nuclei and the nuclei of the oculomotorius nerves were even more atrophied, and yet the root fibers of the oculomotorius nerves on the whole were preserved, although here and there a nerve fiber appeared to be affected. This was a case of primary degeneration of the oculomotorius nuclei in syphilis.

As the individual branches of the oculomotorius often have been separately paralyzed one would a priori expect that partial nuclear disease in such cases would be common, especially in view of the vascular supply, but NONNE as well as WILBRAND and SAENGER have found no such cases with necropsy in the literature. The necropsies have almost all shown disease of the oculomotorius trunk at the base of the brain. UHTHOFF has shown that though the oculomotorius nerve trunk may seem to be uniformly involved in the lesion only functional disturbance of certain muscles may occur, so that from a purely basal lesion only the external or only the internal ocular muscles may be paralyzed, and even that the function of the nerve may be preserved when the microscope has shown the nerve to be affected by the lesion. I have taught for a long time that partial oculomotorius palsy from a basal lesion is explainable on anatomical grounds. The roots of the nerve are not united into a single trunk until they have left the cerebral peduncle. Meningitis may readily therefore cause greater infiltration about certain root bundles than about others.

---

(1) WILBRAND and SAENGER: *Die Neurologie des Auges,* vol. I, 1900, p. 331 and p. 109.

Van Gehuchten (1) recognizes this anatomical fact. He says: « Le nerf oculomoteur commun a son origine apparente sur la face interne du pédoncule cérébral, dans un sillon longitudinal qui sépare ce pédoncule de la substance perforée postérieure ou substance interpédonculaire. Cette origine se fait par un grand nombre de petits filets nerveux qui se réunissent bientôt en un tronc unique ».

Having thus shown by reference to the critical digests made by recent writers on syphilis of the brain that oculomotorius palsy is a common manifestation of this disease the question might arise why anyone should consider an additional case with necropsy worthy of special study. Nonne makes the statement that complete oculomotorius palsy without other functional disturbance of cranial nerves and with only general cerebral symptoms is rare, yet Dineur collected eight cases of isolated unilateral oculomotorius paralysis in syphilis.

Wilbrand and Saenger say that from that course of the arteria peduncularis interna and the arteria nuclei oculomotorii it is understandable why softening in the cerebral peduncle and pons is so often accompanied by nuclear ocular palsy. Necropsy findings of isolated disease of the nuclear region of the oculomotorius from syphilitic endarteritis do not exist, although frequently ocular muscles palsy of nuclear type is seen following syphilis. The explanation is that the etiology of such ocular muscle palsy is recognized and the palsy is cured by proper treatment.

Uhthoff (2), in an article published in 1911, states that oculomotorius palsy without palsy of the body, from lesion in the cerebral peduce, is very rare in cases with necropsy, but has been reported by Achard and Lévi, and Hughlings Jackson, and has always resulted from softening. It is possible that such a condition may occur in commencing or disappearing disease of the cerebral peduncle in cases without necropsy, but the diagnosis of peduncular lesion is uncertain in such cases.

---

(1) Van Gehuchten : *Les Centres Nerveux*, p. 334.

(2) Uhthoff : *Graefe-Saemisch Handbuch der Gesamten Augenheilkunde*, second edition, vol. XI, 2, 1911. p. 636.

UHTHOFF found in the 78 cases of disease of the cerebral peduncle with necropsy he collected partial oculomotorius palsy only in a few cases; he refers to 12 cases. Persistent partial oculomotorius palsy from disease of the cerebral peduncle he regards as relatively infrequent.

The nearest approach to my case is the case reported by ACHARD and LÉVI (1). The lesion in this was unilateral softening, and cut the intrapeduncular fibers of the oculomotorius. These authors mention that an intrapeduncular lesion of this nerve usually is associated with hemiplegia of the opposite side, and the symptom-complex is that of WEBER. An isolated paralysis of this nerve from a lesion within the cerebral peduncle is much more infrequent and they could find no such case in literature.

Their patient was an old woman who was brought to the hospital in coma. She had been seized suddenly with an apopletic stroke, but was not paralyzed. When she recovered consciousness speech was slightly affected although she was not aphasic. The left lower limb was a little rigid but was without true contracture. The reflexes of the upper and lower limbs were not exaggerated. The face was not paralyzed. The left oculomotorius was completely paralysed without implication of other cranial nerves.

A small area of softening was found at the surface and inner part of the left pes pedunculi. It invaded the tegmentum of the peduncle near the aqueduct of SYLVIUS and in this way destroyed the roots of the left oculomotorius.

I am not sure that I have found the case reported by HUGHLINGS JACKSON (2) referred to by several writers.

In one case reported by him complete paralysis of both third nerves developed five months before the patient came under observation. The man stated that these palsies came on suddenly one morning when he was out walking. When admitted there was nothing more than palsies of these nerves.

---

(1) ACHARD and LÉVI : Revue Neurologique, 1901, p. 646.

(2) HUGHLINGS JACKSON : *The Journal of Mental Science*, vol. 20, 1874-1875, p. 235.

His optic disks were normal. He was healthy-looking and went out daily. Later he had two attacks with hemiplegia. At the necropsy both posterior cerebral arteries and the two third nerves were « all fixed to one another by material similar to that thickening the arteries ». The lesion therefore was not intrapeduncular. A part of the cortex of the fissure of SYLVIUS (not the aqueduct) was softened.

A case in which a small tumor within the cerebral peduncle, reported by me in Professor's OBERSTEINER'S Festschrift (1), produced ophthalmoplegia may be mentioned in this connection. The patient, a man 48 years of age, developed paralysis of upward associated movement with loss of convergence. Downward movement was possible, but was difficult. Lateral associated movements at first were preserved. The speech was bulbar in character, and swallowing was difficult. Saliva dribbled from the mouth. Ataxia of station was present. About three months later associated lateral and downward ocular movements became affected. Mentality was poor, speech became more indistinct, and deglutition difficult. Associated upward movement was still lost, and all other associated ocular movements were much impaired. The upper limbs were slightly ataxic. The gait was fair but frequently when he attempted to sit down he would lose his balance and fall backward.

An alveolar sarcoma was found which in its greatest development measured only 5 mm. dorso-ventrally and barely 2 mm. in width in frontal section. The lowest portion of this tumor was seen in the right peduncle where the fibers of the oculomotorius were leaving the nucleus, and some of the fibers of the right oculomotorius passed through it. It was near the right oculomotorius nucleus. The tumor did not extend further forward than the posterior part of the third ventricle. I have had another similar case of oculomotorius palsy from tumor of the tegmentum of the cerebral peduncle, without paralysis of the body or implication of other cranial nerves.

This case of partial ophthalmoplegia from a small intrapeduncular lesion near the nucleus of the right oculomotorius was one without softening. It is not included in UHTHOFF's collection of reported cases of intrapeduncular lesions.

---

(1) Obersteiner's Arbeiten vol. XV, part I, p. 362.

It is important to determine the vascular supply of the oculomotorius nuclei and the manner in which thrombosis of this part of the brain may occur.

VAN GEHUCHTEN (1) gives the supply as follows :

Sur la face antérieure du cerveau moyen, on trouve des *artères médianes*, des *artères radiculaires* et des *artères accessoires.*

Les *artères médianes* naissent de la partie supérieure du tronc basilaire et se comportent comme les artères médianes de la protubérance annulaire et de la moelle allongée. Elles se dirigent directement en arrière, traversent la substance perforée postérieure, qui occupe le fond de l'espace interpédonculaire, et se terminent dans les deux masses grises situées au-devant et un peu en dehors de l'aqueduc de SYLVIUS : les noyaux d'origine du nerf pathétique et du nerf oculo-moteur commun.

Les *artères radiculaires* naissent du tronc basilaire ou de l'une de ses branches collatérales. Elles se dirigent en dehors vers le nerf pathétique et vers le nerf de la troisième paire et se bifurquent alors en une branche périphérique et une branche centrale. La branche périphérique se résout bientôt en un réseau capillaire entre les fibres constitutives du nerf. La branche centrale accompagne les fibres radiculaires du nerf jusqu'au niveau de la masse grise qui leur donne origine et là elle se résout en un réseau capillaire. Le réseau capillaire artériel de chacune de ces masses grises provient donc à la fois et de l'artère médiane voisine et de l'artère radiculaire.

Les *artères accessoires* naissent des artères cérébrales postérieures, pénètrent directement dans le pied du pédoncule cérébral (*artères pédonculaires*) et s'y terminent dans la substance noire de SOEMMERING.

Toutes ces artères appartiennent au groupe des *artères terminales.*

A detailed description is given also by WILBRAND and SAENGER :

---

(1) VAN GEHUCHTEN : *Les Centres Nerveux cérébro-spinaux.* 1908, p. 341.

« Das Kern-und Wurzelgebiet des Oculomotorius wird von der Arteria basilaris aus mit Blut versorgt. Dieselbe theilt sich am Vorderende des Pons in die beiden Arteriae cerebri posteriores profundae ». « An der Stelle der Bifurkation der Arteria basilaris, am vorderen Rand der Brücke, gehen nun in der Richtung nach vorn und entsprechend der Substantia perforata posterior einige kleinere Arterien ab, welche die Ernährung des grösten Theiles der Hirnschenkel und des hinteren Abschnittes der Thalami optici besorgen. Die erste von diesen Arterien, die Arteria peduncularis interna, versorgt den inneren Abschnitt des Hirnschenkelfusses, hauptsächlich die Austrittsstelle der Wurzelfasern des Nervus oculomotorius ; die zweite, Arteria nuclei oculomotorii, ernährt sämmtliche Kerne dieses Nervenpaares im ganzen Verlaufe der Haube und des hinteren Abschnittes des Boden des III Ventrikels. Die dritte, Arteria optica interna et posterior, geht weit nach vorn und speist den hinteren Abschnitt des Thalamus opticus, das Pulvinar. Das vierte Gefäss endlich, die Arteria pedunculo-gemina, fuhrt Blut zu den vorderen Vierhügeln und bildet in Gegensatz zu allen übrigen Gefässen dieser Gruppe keine Endarterien, sondern geht vielfach anastomotische Verbindungen mit den Gefässen der benachbarten Arterien ein (Rossolimo).

Eine Erkrankung der Basilararterie, zumal an ihrer Bifurkationsstelle, wird daher sehr leicht Ernährungsstorungen im Gebiete der Oculomotoriuskernregion zur Folge haben können ».

In another place Wildbrand and Saenger say : « Wir wissen ferner, dass der Oculomotoriuskern selbst wiederum von zwei getrennten Gefässgebieten ernährt wird, indem der vordere Theil von der Arteria cerebri posterior, der hintere von der Arteria basilaris sein Blut erhält (Knies), so dass sehr wohl z. B. ein Erkrankungsherd im vorderen Abschnitt des Oculomotoriuskerns, ein anderer im Trochleariskern, und ein dritter in Abducenskern Zerstörungen angerichtet haben kann, ohne dass der dazwischen liegende hintere Theil des Oculomotoriuskerns affizirt zu sein braucht. Für die Thatsache, dass dabei die vom Oculomotorius versorgten Mukseln ganz besonders häufig in ihrer Funktion beeinträchtigt erscheinen, macht Shimamura folgende Gründe geltend. Erwägt man, dass der Oculomotoriuskern ungefähr an derjenigen Stelle des Gehirns liegt, wo die von der Carotis einerseits und der Vertebralis anderseits kommenden Blutwellen auf einander stossen, bedenkt man ferner, dass alle die Gefässe, welche sich schliesslich im Oculomotoriuskern in Endäste auflösen, in fast senkrechtem Verlauf von der Basis nach der dorsalen Seite aufsteigen, und berücksichtigt man drittens, dass diese Gefässe, wie die Injektionsversuche ergeben haben, Endarterien sind, dass also der Oculomotoriuskern nur von diesen Aesten und keinen anderen sonst sein Blut erhält, so können diese drei ungunstigen Umstände wohl eine genugende Erklärung dafur geben, warum der Oculomotoriuskern so häufig der Sitz von Erkrankungsprozessen ist, und warum eine Reihe von Erkrankungen des Centralnervensystems ihre ersten Zeichen in das Gebiet der vom Oculomotorius versorgten Theile verlegen. Es dürfte bei jeder Störung der Circulation, besonders bei jeder Herabsetzung des Blutdrucks, hier am allerersten eine Blutleere eintreten, welche zuerst zu vorubergehenden, und bei häufiger Wiederholung auch zu andauernden Störungen Veranlassung geben könnte ».

Extracted from the American Journal of the Medical Sciences,
September, 1913, No. 3, vol. cxlvi, p. 406.

THE POLYNEURITIC FORM OF ACUTE POLIOMYELITIS: A CLINICAL AND PATHOLOGIC STUDY.

By S. Leopold, M.D.,
INSTRUCTOR IN NEUROLOGY AND NEUROPATHOLOGY, UNIVERSITY OF PENNSYLVANIA.

The question of the existence of a true polyneuritic form of acute poliomyelitis has never been satisfactorily determined. As far as I am aware no case has ever come to necropsy. Wickman in his article on "Acute Poliomyelitis" in Lewandowsky's *Handbook*, published in 1911, states "that he was unable to find in the literature a report of this type in which the peripheral nerves had been studied." Recently, I had the opportunity of studying a fatal case under the care of Dr. Pitfield.

A. S., female, aged twenty years, was admitted to the hospital on August 17, 1911. Nothing of importance was noted in the family or medical history. Her illness began several days previous to admission with nausea, vomiting, and fever, accompanied by severe pain in the back of the head and neck. A few days later pains and paralysis occurred in both lower limbs, especially the left. Physical examination of the heart and lungs showed no changes. The abdomen was somewhat distended.

The left lower limb was completely and the right lower limb was partially paralyzed, and only the foot could be moved. Pain on pressure over the nerve trunks was noted in both limbs. The left knee-joint was swollen. There was paralysis of the bladder.

No changes were found in the urine. The blood examination showed: Red blood cells, 4,600,000; white blood cells, 7200; hemoglobin, 80. The Wassermann reaction was negative.

August 29. General condition as on admission, save that the patient voids urine voluntarily.

September 18. Pupils equal; reacted to light and accommodation. There were no ocular palsies. The eye-grounds showed no changes. There was no involvement of the cranial nerves. She could move either arm freely, and in all directions. The biceps-jerk was normal. There were no sensory disturbances in the arms or hands. She could move the right foot and leg slightly. The left limb could not be moved at all. Both patellar tendon reflexes were absent. The knee-joints were swollen. Tactile sensation was diminished in both lower limbs. A pin-point was felt over the entire left lower extremity, but not so distinctly over the right lower limb above the knee. There was marked tenderness over the nerve trunks in the lower limbs.

September 24. The urine showed casts and albumin. The condition of the patient remained the same, except that the temperature rose on the thirty-third day, and remained between 102° and 104° until death.

History. A young adult female, without any previous illness, was suddenly seized with vomiting, fever, pain in the head and back, followed in several days by pain and paralysis in the lower limbs, together with paralysis of the bladder. The important physical symptoms were loss of patellar tendon reflexes and unsymmetric paralysis of both lower limbs, with partial areas of hypesthesia. There was bladder paralysis. In addition there was marked tenderness on pressure over the nerve trunks in the paralyzed extremities, which persisted until death, two months after the onset.

At necropsy the heart, lungs, spleen, and liver showed no changes. The kidneys showed cloudy swelling. A few punctate hemorrhages were seen in the visceral pericardium.

No exudate was noted in the meninges of the brain or spinal cord. The vessels were injected. The cord in the lumbar region was somewhat softer than in the other portions, and on section showed a reddish-gray area in both anterior horns.

Microscopic sections were made from various levels of the spinal cord and medulla oblongata. These sections were stained with thionin hemalum and acid fuchsin, by Weigert's stain, and by the Marchi stain.

Only one peripheral nerve and portion of one muscle were obtained and these were from the anterior aspect of the right thigh, near Poupart's ligament. A portion of the nerve was placed immediately in osmic acid and teased after twenty-four hours. Another portion was placed in Müller's fluid and studied by the Weigert, hemalum, and acid fuchsin stains. There was no evidence of an acute or subacute inflammatory process, and no distinct degenerative changes were noted. The hemalum-acid-fuchsin stain showed slight increase in the connective tissue. There was no round-celled infiltration. The osmic acid showed slight swelling of the fibers, some granulation of the myelin, but no breaking up into droplets. Most of the fibers were normal with Weigert's stain.

The section of muscle showed no round-celled infiltration, no swelling or atrophy of fibers. The striations could be seen, but were not sharply defined.

In the medulla oblongata no changes were noted.

The cervical and thoracic areas of the spinal cord showed no changes.

In the lumbar area stained with the hemalum-acid-fuchsin the meninges showed an intense round-celled infiltration.

The pial vessels were dilated and intensely engorged with blood. Around and within the anterior and posterior roots the round-celled infiltration was intense, and the capillaries were intensely dilated.

The anterior horns showed the characteristic picture of the subacute stage of an acute poliomyelitis.

The left anterior horn was more involved than the right. The capillaries were injected, the round-celled infiltration was marked, and the cells showing a clear protoplasmic area around the nucleus.

Many lymphoid cells, a few connective-tissue and polymorphonuclear cells were also seen. The ganglion cells had practically disappeared as only a few were seen, and those remaining were atrophied. With the Nissl stain the disappearance of the ganglion cells and the varying grades of degeneration of those remaining were most marked.

No changes were seen in the posterior horns or in the white matter. The Weigert stain showed no sclerosis.

These changes were noted in the second, third, fourth, and fifth lumbar segments.

PATHOLOGIC HISTORY. The changes found in the lumbar region of the spinal cord were those of an acute poliomyelitis in the reparative stage.

The anterior horns were practically destroyed. Intense inflammation was noted in the meninges and around the posterior roots. Most interesting was the exudate and inflammation of the spinal roots.

The peripheral nerve showed no evidence of a primary neuritis, and the changes present were probably secondary in character.

The diagnosis of this case was difficult. The possibility of a true multiple neuritis had to be excluded. Pain on pressure over the nerve trunks, which is emphasized so much in text-books, could not be considered as a differential symptom.

True multiple neuritis develops slowly, fever is usually absent, special groups of muscles are affected, and the distribution is more symmetric. The absence of sore throat and any history of diphtheria precluded a diphtheritis neuritis, the paralysis in this also occurs much later than the throat disorder; a neuritis of other type, could not with certainty be excluded. The diagnosis of the polyneuritic form of acute poliomyelitis was substantiated by the presence of several other cases in the same vicinity, the unsymmetric involvement of the paralysis, and the absence of cranial nerve involvement, though there are cases of multiple neuritis in which only the spinal nerves are affected, and cases of acute poliomyelitis have been reported with cranial nerve paralysis.

The resemblance of multiple neuritis to poliomyelitis has been known ever since Leyden recognized a clinical relationship.

This similarity in the clinical pictures of the two diseases was recognized in the chronic form of the spinal disease, but recent studies have established the resemblance to acute poliomyelitis.

Cases were described clinically by Strümpell and Barthelmes in 1900. Wickman in 1905 reported a few cases, and in the epidemics observed by Hartman, Foerster, Müller, Mills, and Spiller this form has been recognized. In Foerster's cases everyone showed pain and tenderness on pressure over the nerve trunks, which continued for months. At least clinically there seems to be no question of a polyneuritic form. These cases are remarkable in the prominent part played by the pain over the nerve trunk. After a shorter or longer interval, varying from days to several months, this pain disappears. These cases may recover completely or show permanent paralysis; in rare instances they are fatal.

Those cases in which the pain resolves itself into a hyperesthesia of the skin or on movement of the joints or limbs should not be included in the polyneuritic form. Many cases of acute poliomyelitis may show this at the onset of the disease.

In some of the polyneuritic forms, objective sensory symptoms may be present, such as hypesthesia and disturbances of pain and temperature.

Pathologic evidence of this form is wanting. The peripheral nerves have never been examined in any such case. Cases of acute and subacute poliomyelitis not of the neuritic type have been reported in which the peripheral nerves were studied. In the cases of Redlich, Monkberg, Mott, and others the nerves were found degenerated. This degeneration could be attributed to a secondary change, owing to the long duration of the cases (from thirteen days to several months).

Harbitz and Schell's cases of acute poliomyelitis, with pain, do not answer to the polyneuritic type, though their findings are of interest.

In their Case VI, a boy, aged six years, had headache, pain in the neck, and pain in the arms. Examination of the brachial plexus and the vagus nerve showed no inflammation or degeneration.

In their Case VIII, one of poliomyelitis, with bulbar symptoms, in which pain played a prominent part at the onset, the vagus, phrenic, and cervical plexus showed no changes.

Their Case X showed an acute onset, with headache, fever, pain in the limbs, and bladder paralysis. No changes were noted in the peripheral nerves.

Wickman believes that the peripheral nerves are never involved and that the pain is due to the meninges, while Raymond held the view that the whole peripheral neuron is affected.

Strümpell, while admitting the involvement of the peripheral neuron, believes that the two diseases are distinct, in that in the spinal type we are dealing with a focal lesion, while in the neuritic form we have the effect of a toxic disturbance in the general circulation.

The pathologic evidence was wanting of any primary degeneration of the peripheral nerve in my case. There were no inflammatory changes. That only one nerve was studied renders this evidence insufficient, but it is reasonable to assume that even this should have shown some neuritis.

Changes may, however, be absent even in true neuritis. Spiller has examined the nerves in different cases of peripheral neuritis, and in some instances he has failed to find any change. This he attributes to the unequal distribution of the inflammatory process, though clinically in his cases the pain was felt along the entire nerve. The more central portions of a nerve may escape when the peripheral portions are diseased. Swelling of the nerve at one portion would produce pain throughout, yet the nerve above the swelling might not show any changes.

Pathologic evidence was not lacking to explain the pain in the case reported. The intense inflammation in the meninges, quite marked around the posterior roots, and the swelling of these roots, on account of the exudation of the round cells within them, as well as the intense engorgement of their vessels, might well account for the pain. The tenderness along the nerve trunks could easily result from this central irritation. In this connection it would be interesting to know whether in other diseases of the spinal cord such as tumor, etc., in which the spinal roots are irritated, tenderness could be elicited along the nerve trunks. That it occurs usually in acute meningitis is well known.

45

44.9

Muscle Tonicity, Emotional Expression, and the Cerebral Tonectic Apparatus.

Considered especially with reference to a case of bilateral caudato-lenticular degeneration.

By **Charles K. Mills, M. D.,**
Professor of Neurology in the University of Pennsylvania.

My object in this paper is to discuss some of the problems connected with affections exhibiting abnormal muscle tonicity and involuntary emotional expression from disease of the striatum and frontal cortex. The paper is based in large part on the detailed study of a case under my care in the Philadelphia General Hospital at recurring periods during seven years. It is hoped that the record here presented will add to our knowledge of the symptomatology of lesions of the striatum.

Onset and general course of the case.

T. W., 37 years old, was admitted to the Philadelphia General Hospital on April 8, 1906. He died November 16, 1913. During this long period he was a patient in the Men's Nervous Ward.

When admitted to hospital he gave the history of having drunk wood alcohol for two years and also of syphilitic infection in 1903.

Several months before his admission he had suddenly lost power in the right arm, leg and right side of the face. In a few days, according to his wife, his loss of power apparently completely disappeared. On the day before his admission, namely on April 7, 1906, he was paralyzed in a similar way on the left side of the body, but power partly returned on this side also. At the time of this second attack he had pain in the left side of his head after the oncoming of which he could not move the left eye outwards.

On the day of admission examination showed that the left pupil was about normal in size, the right slightly larger and both reacted to light. Extra-ocular movements were well performed except external rotation of the left eye. On this side he could not open the lids widely and could not close them tightly. When the left eye was at rest internal strabismus was present. There was no hemianopsia. No ophthalmoscopic examination was made at this time, but later examinations showed that papilloedema and atrophy were not present. The left naso-labial fold was not quite so marked as the right and the patient was not able to smile or to draw up either side of his mouth.

This patient's condition remained much the same during 1907, 1908, and 1909. He gradually failed, however, his failure becoming more marked in 1910 and 1911. His main symptoms did not change greatly during the entire period of his stay in the hospital except for the coming on of more complete helplessness, of more marked contractures and rigidity and of a mental condition in which he gradually responded less and less to his surroundings.

Pyramidal symptoms.

The patient had a few symptoms which were clearly due to lesion of the posterior limb of the capsule on each side. It will be seen later that on the left side a sclerosed area only about 2 mm. in width was present exactly at the knee of the capsule. The weakness observable in the movements of the lower face on the opposite side was doubtless due to this lesion. On the right side, it will also be seen that an area of complete sclerosis occoupied one-third or more of the anterior part of the posterior limb of the internal capsule. Paresis of jaw movements on the left and of the left side of the face were referable to this lesion. Loss of power in the left upper extremity due to lesion of the posterior limb of the capsule on the right may have been masked and altered by the symptoms of extensive lesions of the striatum and anterior limb of the capsule. Notes were made at various times regarding the paretic condition of the facial and maxillary musculature.

Extra-pyramidal symptoms.

Among the most striking manifestations in this case were the attacks of involuntary painful emotional expression. The symptom was at its height during 1907, 1908, 1909, and 1910. During the last three to four years before his death the attacks became less frequent and less marked.

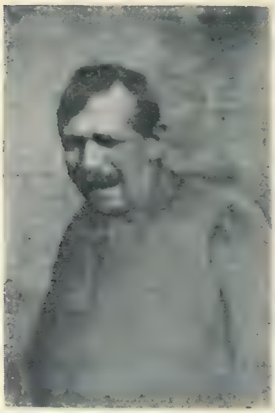


Fig. 1. Expression in involuntary howling

When asked to open his mouth or questioned in any way, he at once burst into an involuntary distressing cry which increased in intensity becoming a prolonged howl. The expression of his face (Fig. 1) during this period was one of pain, but the existence of true emotion was doubtful.

Another record states that the upper and lower face muscles were drawn into the position of crying, the jaw being dropped and the mouth opened. Lachrymation did not occur; the seizures were never alternated with paroxysms of spasmodic smiling or laughing and the latter never occurred separately. He could extend his tongue without howling, but he began to howl as soon as he began to talk.

From the time of my first examination in 1906 hypertonicity was one of the most marked manifestations in the case. Its presence is illustrated in some of the notes which have been made regarding other phenomena, as when discussing station, gait, and special attitudes, paresis or paralysis, tremor, etc. The hypertonicity affected both the upper and lower extremities and the trunk (Figs. 2 and 3). The limbs were held in various stiff, rigid, awkward positions, but could be straightened, even by the patient himself with effort, for several years. Eventually he became a stiff, ungainly mass, always confined to his bed. His lower extremities in these late periods exhibited a spastic tendency in the entire musculature, which resulted in the limbs retaining at times an almost straight position. On October 26, 1908 the following notes were made: „The arms have a tendency to assume various positions. At times they seem to be spastic, or rather pseudo-spastic, but usually it is a more or less toneless fixed position. His grip in each hand is poor, although he paroxysmally grips harder.“

The legs at this time were extremely hypertonic, but had not become fixed in a position of permanent contracture.

When he was first admitted in 1906 he was able to stand and to walk a little, but both station and gait were very peculiar. He could stand with assistance for a short time until late in 1908. In January, 1907 it was noted that on being urged to get up he did so and walked a few steps. The body when walking was held rigidly and somewhat bent forward and somewhat to the right. His gait was very rigid; he walked on the fore part of his foot and toes and took very short steps (Fig. 2).

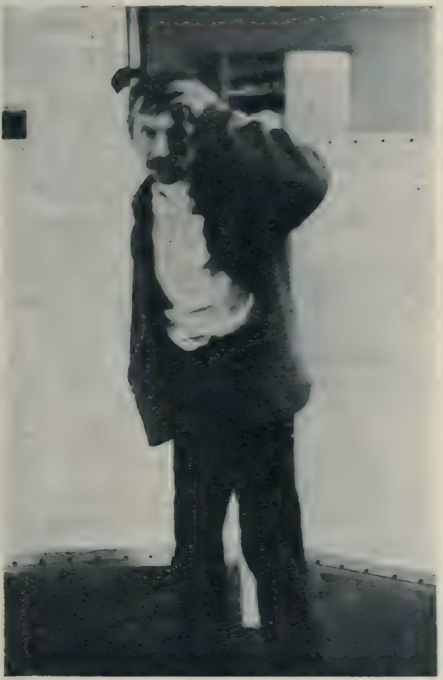


Fig. 2. Station in attempting to walk, showing hypertonicity and forced attitude with trend to right.

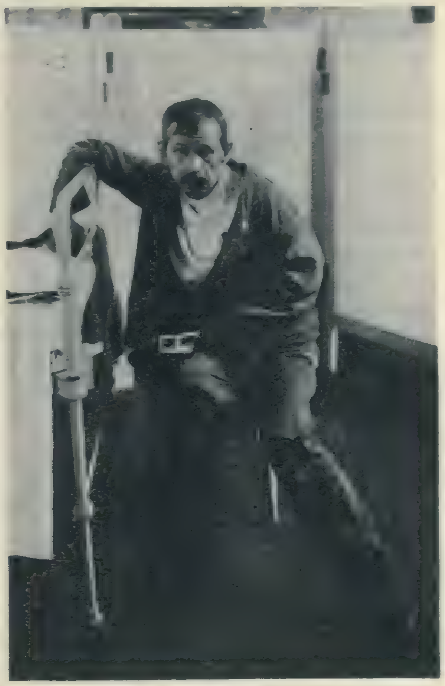


Fig. 3. Facial expression and trend to the right in sitting.

With regard to his ability to stand the note was made on October 26, 1908, that when placed upon his feet, with someone supporting him from behind, in trying to stand the left foot was flat on the floor, the right leg flexed somewhat at the knee and hip, with the toes of the right foot resting on the floor. In trying to walk he was unable to advance the right leg and foot, or at least he did not do this. He advanced the left leg, and with this semiflexed he rested on the toes.

Even in sitting, so long as he was able to do this, he showed a tendency to special attitudes, the head and trunk inclining en masse to the right (Fig. 3).

Some of the phenomena exhibited by the patient may have belonged under the head of perseveration, the symptom which Wilson has described in cases of tumor of the intermedio-precentral region. He had some tendency to what might be termed paroxysmal gripping as stated when discussing hypertonicity. The accidental discovery of a method of managing his upper extremities when arranging his bed and at other times may perhaps illustrate the perseveration.

His arms were extended back of his head and made to clasp the uprights at the head of his bed. When this was done, he would continue them in this position for an indefinite time, an hour or more, until they were unclasped by the nurse or attendant.

Tremor was a marked manifestation. It never affected his lower extremities, but was exhibited in movements of the head and upper extremities. In January 1907 the note was made that in sitting sometimes his head was held over the edge of the bed. While in this position he was either sleeping (which he did most of the time), or he was constantly moving his head and upper limbs. He turned his head from side to side and at the same time he rubbed his hand on the bed, repeatedly with the same movement. Sometimes it was like the movement of pill rolling, but this might change to some other movement. The repeated movements occurred also in the left hand.

On October 26, 1908 I made the following among other records: „The finger-to-nose test is tried with each hand. He evidently understands the directions and tries to carry out the test. On approaching the finger to the nose it begins to hover and tremble. He is distinctly ataxic or uncertain in the movements.“

I have already described what was probably a pyramidal or voluntary motor paresis, this having been chiefly observed in movements of the face, tongue, and jaw. In all parts of the body, however, there was some diminution of power associated with the hypertonicity and other symptoms. Even when his spasticity and contractures were somewhat advanced he could still use his limbs in response to urging or in resisting passive movements, but he did this with difficulty and with little energy.

The note was made October 26, 1908 that lying in bed with the legs drawn up the patient was told to straighten them. He evidently understood the order and tried to push down first one limb and then the other. He managed to get them nearly straight with many efforts. He did not seem to know just exactly how to carry out the order or could not keep up a continued and well directed effort.

Numerous observations were made of the patient's speech. He had a form of dysarthria associated with what might be termed a sort of neurasthenia of speech. On January 29, 1907 record was made that the speech was indistinct, the voice was low and without inflection. The speech was not bulbar, but was rather that of one who does not want to make an effort or is too tired to speak. Although low and feeble, it could be understood on close attention. When asked why he moved his head the patient said, „Habit I suppose“. He also said, „I am better than I have been for some time“. He made other replies in short sentences indicating that he knew what was asked of him and that he could answer in intelligible language, although his speech was limited. After speaking a little his voice became more indistinct with some apparent mental confusion.

On October 27, 1908 the following interesting notes as to this speech were made by Dr. WEISENBURG: „His voice is low. He pronounces fairly well. *B* and *p* are pronounced well; on *r* he does not get the trill; he does not pronounce *k* at all; he can say *s* but cannot prolong the expiratory sound; he can say *m* or at least can start saying it; and he can say *n* in the same way; he can say *f* and also *g*; when he tries to pronounce *Georgia* or *eggs* he slurs over them.“

As time went on the patient talked less and less during efforts made to test him. His spontaneous speech, always extremely limited, disappeared.

From an early period after his admission to the hospital the patient had difficulty both in mastication and in deglutition. Most of the time he was able to take only soft diet, and had to be carefully fed. On September 27, 1908 it was noted that he would not attempt to swallow until his mouth was full, and then he would choke in swallowing. If given a little food at a time he could be made to chew and swallow.

From first to last I found it difficult to measure with any approach to exactness this patient's mentality. Members of the staff who observed him and in some cases made notes as to their impression differed somewhat in their conclusions. All, however, seemed to believe that some mental reduction was present. Even notes observations made as early as 1906 and 1907 record this impression. On January 29, 1907, it was recorded that in appearance he reminded one of the bestial, as if there was little or no intelligence left. He paid no attention to his excretions. When asked to show his tongue or move his limbs, he did so in a dull hesitating manner as if he only partially comprehended what was asked of him. His memory was undoubtedly affected and he showed a tendency at times to memorial fabrications. He became duller and more confused and inattentive as time went on.

In spite of the above facts it was remarkable how much he seemed to understand of what was said to him and so far as could be determined by his looks and the movements of his head and eyes of what was going on around him. Vasomotor and other symptoms referable to the autonomic nervous system were present. In his attacks of involuntary painful emotional expression the patient's face usually became very turgid, and a somewhat congested appearance of the face was present at all times. On September 27, 1908 Dr. WEISENBURG noted that involuntary crying was frequent. The patient nearly always started with a cry and ended with a howl and when he did so his whole face took part in the movement; the face became red and his eyes had the appearance of crying although no tears were shed. In about one-half a minute the face and eyes cleared up. On October 26, 1908, I noted that the man's face when at rest was always redder than normal, and that during the attacks of howling the face was intensely flushed.

Drooling of saliva was a troublesome, and after the first year a frequent symptom. The patient had a tendency, almost continuous or very frequently recurring, to grind his teeth, this with the rest of his facial expression giving his countenance a distorted and painful appearance. On account of this tendency the nurse often placed a towel between his teeth.

The patient suffered much from sexual or genital irritation. At least, he showed a tendency to handle himself in an unpleasant way, because of which it was necessary at times to confine his hands.

Throughout his illness he had what might be termed an irregular incontinence of urine and feces, often soiling himself if not closely watched, although at other times he gave notice of his necessity to evacuate bladder or bowels. During the last three years he paid little or no attention to his evacuations.

No observations were made with regard to sweating or to the condition of the hairs.

The temperature, pulse and respiration were taken from the time of the patient's admission, April 8, 1906, for about four months. The results were so nearly uniform that they can be summarized in a few words. For fully three-fourths of the time the temperature ranged between half a degree and a degree (Fahrenheit) below normal and occasionally fell as low as 97. It ran generally between 97.5 and 98 degrees. The pulse was generally high, usually between 90 and 110, although occasionally it went above or below these figures. The respirations usually ranged between 20 and 25.

Negative and miscellaneous symptoms.

Sensation was normal in all its forms. The deep reflexes both in the lower and upper extremities were very marked, but no Babinski response was present. Foot clonus and patellar clonus were absent. The abdominal reflex was present. Numerous observations confirmatory of these facts were made from 1906 to 1911.

Nystagmus was absent. No disorder of ocular movements was present except left abducens paralysis for a short time early in the history of the case. This disappeared and was probably due to some infiltration of syphilitic origin. Papilloedema was absent. The pupils varied a little in size, but the pupillary reflexes were normal.

The patient gradually became very thin and weak. Fibrillary tremors were absent. Atrophy in the true sense was not present.

Gastro-intestinal symptoms were absent. He never exhibited any jaundice. The conditions of the thoracic and abdominal viscera found at the necropsy were probably of late origin. At one time the patient had an ischio-rectal abscess which was relieved by surgical treatment and at another he suffered for a short time from acute conjunctivitis.

Somnolence was a marked symptom especially observed in 1907 and 1908. The following is a citation from the hospital notes.

January 29, 1907: „He falls to asleep very easily, in fact while he was being examined and after answering a question he suddenly shut his eyes, his head drooped over and he began to sleep. During sleep, the face assumed an expression of care, that is, there was puckering of the muscles of the brow and eyes. While sleeping the head drooped farther and farther and there was dribbling of saliva. The limbs remained constantly in the same position and there was no tremor. The nurses state that he sleeps almost constantly and he has to be wakened, this sometimes being done with difficulty. Sometimes while he is being fed he falls asleep.“

General necropsy.

The necropsy was held eighteen hours after death. The cardiac musculature was pale and rather flabby. The wall of the left ventricle was 1·8 cm. in thickness. The aortic and mitral leaflets showed no abnormal changes. The right ventricle showed slight adhesions to the tricusped leaflets. In the mediastinal space was found a large firm irregular node which on section showed a number of small irregular partly caseous and partly calcified areas. The upper lobe of the left lung was of a light gray color; the lower lobe was of a dark red color. On section the upper lobe crepitated throughout, while the lower lobe showed an irregular triangular area with its greatest diameter just underneath the pleura. In the vessel leading to this area was a dark red thrombus. The upper lobe of the right lung was of a light grayish color, while the lower lobe showed on section a number of small elevated grayish areas which on being excised sank in water. The capsule of the spleen was thickened. The aorta showed a number of small elevated grayish patches principally around the orifices of the vertebral vessels. The capsule of the liver was smooth and tense. The edges were sharp. Cut surfaces were cloudy, granular and congested. The gall bladder was distended and contained four large irregular biliary calculi. The left kidney showed its cut surface to be extremely congested. The capsule stripped bodily leaving a smooth cortical surface. The left supra-renal body showed no macroscopic change. The right kidney and adrenals resembled the left. Negative and unimportant findings are omitted. The brain was extremely edematous. The lateral ventricles were bulging and enlarged on account of the excessive amount of fluid. External examination showed no gross evidence of a lesion except that at the base of both lateral ventricles the brain substance appeared to be softened and of brownish color.

Examination of the brain.

The brain and spinal cord were sent to the Laboratory of Neuropathology of the University of Pennsylvania and were examined by my colleague, Dr. William G. Spiller, Professor of Neuropathology in the University, to whom I am indebted for the subjoined report.

The specimens were placed in a five per cent. solution of formalin and at the end of a week two large sections of the brain were made.

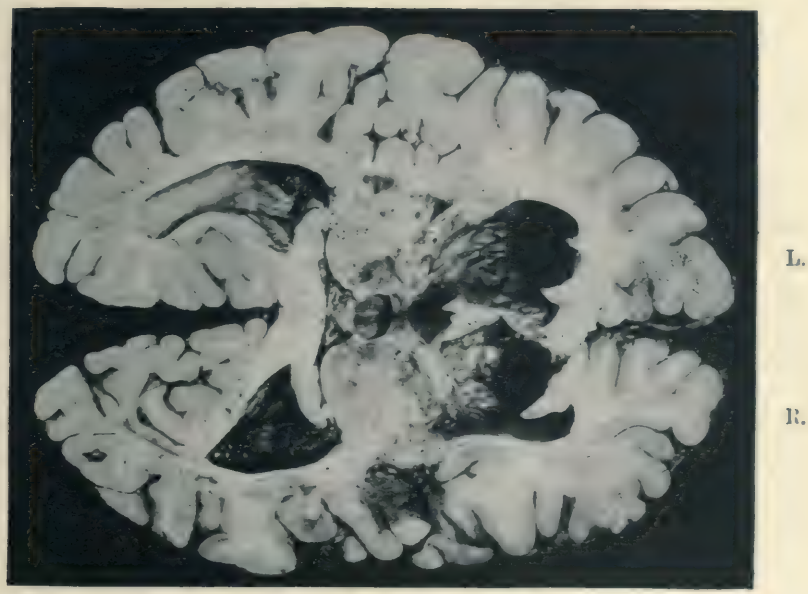


Fig. 4. Horizontal section of brain just below the level of the callosum, showing the destructive lesion of the caudatum and lenticula on the right and of the caudatum and a small area of the lenticula on the left.

A horizontal section just below the callosum passing through the upper part of the thalamus showed an old area of sclerosis and softening in the lower part of the right ascending parietal convolution, the posterior half or a little more of the island of Reil and the tip of the first temporal convolution. The anterior horn of each lateral ventricle was much enlarged. The head of the right caudatum and the anterior limb of the right internal capsule had disappeared. The head of the left caudatum was much atrophied. The anterior limb of the left internal capsule was intact. A second transverse cut was made half an inch below the first. At this level there was an area of sclerosis on each side, nearly symmetrical, involving the anterior part of each lenticula. The posterior horns of the lateral ventricles were not distinctly enlarged.[1]

---

[1] For the photographs reproduced in Figs. 4 and 5 I am indebted to Dr. Alfred Reginald Allen of the Neurological Staff.

The basal ganglia and pons were cut in microcospical serial sections. The anterior third or half of the right lenticula, including the pallidum and putamen, was replaced by sclerotic tissue as was also the anterior third or half of the posterior limb of the internal capsule. The ansa lenticularis was greatly degenerated. The right corpus Luysii was about one-third smaller than the left corpus Luysii, as was also the ruber on this side. Sections from the higher levels of the basal ganglia on the left showed that the anterior limb of the internal capsule was intact, but a small area of the posterior limb, 2 mm. in extent exactly at the knee of the capsule, was degenerated. The head of the caudatum was about half the normal width. At low levels where the ruber was well developed the area of softening and sclerosis cut the few remaining fibers of the anterior limb of the internal capsule and the extreme anterior and inner part of the lenticula, implicating the ansa lenticularis.

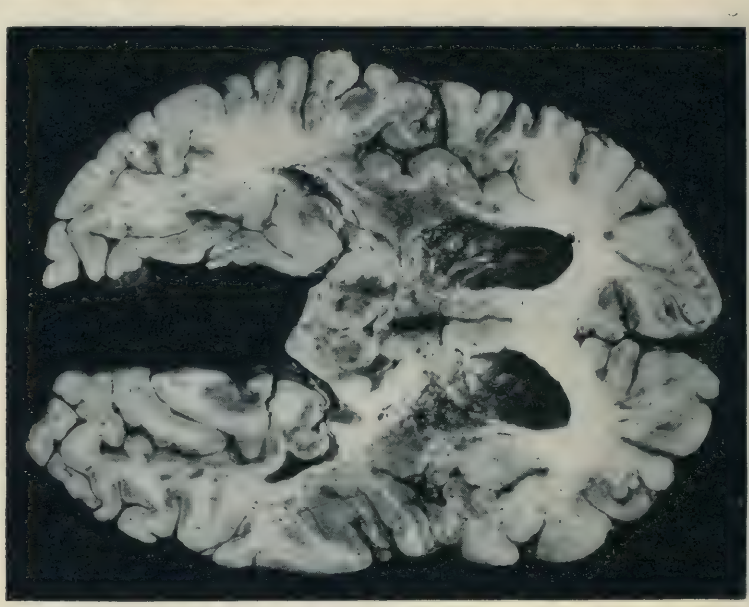


Fig. 5. Horizontal section of the brain about half an inch below the level of the callosum, showing on the right destructive lesions of the caudatum, lenticula and of the anterior third of the posterior limb of the capsule; on the left, destructive lesions of the caudatum and of the anterior portion of the lenticula.

Sections from this level stained with haemalaum and acid fuchsin showed much calcification of the small vessels near and in the degenerated area.

The right anterior pyramid was distinctly but not intensely degenerated. The left anterior pyramid was slightly degenerated. The left crossed pyramidal tract in the cervical region of the cord was moderately degenerated; the right crossed pyramidal tract appeared nearly normal.

Distinct proliferation of the intima of the basal vessels, and considerable lymphocitic infiltration of the pia were present.

Comments on the symptomatology.

The pyramidal symptoms in this case represent a small percentage of the entire symptomatology, probably less than ten per cent. When this is said, however, it will be understood that the phenomena of muscle tonicity which constitute the most striking features of the case are expressed in modifications of voluntary and autonomic motility. In other words, the extra-pyramidal apparatus and its phenomena are supplemental or accessory to the voluntary motor and autonomic motor systems. The pyramidal symptoms on the left side of the body were those due to destructive involvement on the right side of about one-third of the posterior limb of the internal capsule. These were chiefly paresis of cerebral type in the distribution of the left seventh nerve and of the motor branch of the fifth and probably limited paresis in the left upper extremity. Slight paresis in the distribution of the lower branch of the seventh nerve was due to the very limited lesion of the capsule on the left.

In the summing up, the symptoms due to the limited sclerosis of the inferior parietal lobule, the posterior half of the insula and the tip of the temporal lobe can probably be disregarded.

The motor extra-pyramidal symptoms will be seen on examination to have for their keynote muscular hypertonicity. Little or no discussion of the question of hypertonicity is necessary in considering such evidently hypertonic phenomena as spasticity, rigidity, contractures, perseveration and forced attitudes, in which hypertonicity is evident. Involuntary painful emotional expression, so dramatic a feature in this case, like involuntary agreeable emotional expression so often observed in lenticular degeneration, is also a symptom of hypertonicity, at least in large part. The expressions „spasmodic laughing“ and „spasmodic weeping“ as descriptive of these cases are correctly used. Tremor is a phenomen of unstable or irregular tonicity. True impairment of power, while unquestionably present was hard to measure and to interpret. Perhaps it might be regarded as an affective motor rather than a voluntary motor paresis, the withdrawal of emoto-tonic excitations lowering the energy and interfering with synergy of movements, as will appear more clearly when the cerebral-tonectic apparatus is discussed. The dysarthric and dysphagic disturbances were in part, at least, manifestations of hypertonicity. I could not, however, convince myself that these symptoms could be fully explained on Wilson's idea that they were entirely spasmodic. It must be remembered that the pyramidal lesions in this case affected movements which are concerned with speech, mastication and swallowing. The peculiar manner of the speech of the patient reminded me sometimes of Wilson's description of the speech of one of his patients recalling that of a ventriloquist. Voluntary motor paresis, affective motor paresis and hypertonicity may all have entered into the explanation of the symptoms. Movements concerned in speech and deglutition, and indeed all the skeletal movements performed by the patient, might be described, to use a colloquial expression, as „lacking steam“.

I have already sufficiently discussed the question of mentality and also in presenting them the sensory, reflex and other negative signs and symptoms. Unlike the case in which large destructive lesions have been limited to the lenticula on one or both sides, vaso-motor, secretory, respiratory and other abnormal phenomena referable to the autonomic or sympathetic nervous system were exhibited by this patient. It is interesting to note that in connection with painful emotion and the painful expression of emotion by facial and limb movements, the phenomena of the autonomic nervous system such as vasomotor, secretory, pilatory and glandular manifestations play a prominent role. The somnolence so strikingly exhibited by this patient over a long period is difficult to explain, unless it was due to syphilemia or some toxic condition produced by disorders of the gastro-intestinal tract and abdominal viscera. I have, howewer, seen this symptom in cases of tumor of the frontal lobes.

The cerebral tonectic apparatus.

A comprehensive recall of the symptomatology of the case just presented shows that hypertonicity and painful emotional expression, the latter being largely due to abnormal muscle tone, are the chief features which invite discussion. The abnormal phenomena exhibited are referable to lesions of what might be termed the affective motor in contradistinction to the voluntary motor apparatus. As these phenomena are chiefly concerned with cerebral tone, the term „tonectic“ has been chosen as the most descriptive designation.

A degree of atonia is sometimes present as a result of cerebellar lesions, but this fact can be explained without assuming that tonus is a fundamental cerebellar product, as was held by Luciani. The three essential qualities of motor innervation are energy, tone, and synergy. Tone is necessary for the rhythmetization both of voluntary and involuntary movement; synergy in order that toned movements shall be grouped for the performance of acts which require special adjustments. Tone integrated in the striatum unites with cerebellar synergy through the intermediation of the cerebello-rubro-thalamo-cortical apparatus and the cerebello-rubro-spinal apparatus. If the cerebellar influence is withdrawn, asynergic movement is the consequence. If the tonectic excitations from the striatum are withheld, hypertonicity and involuntary movement result.

Tone at least in the human being, is not a spinal or cerebellar attribute, but belongs to the cerebral cortex, becoming more cortical, as Crocq has shown, as we ascend from animals lower in the scale, the frog, the dog and the ape, for instance, to man. That the cortex of the cerebrum is the seat of tone is not a new idea. It has been held by many, the views of those expressing the theory however being somewhat diverse. In most instances the pyramidal motor cortex seems to be regarded as the rendezvous and storehouse of the tone derived from sensation and idea. The cerebral tonectic apparatus, I believe, is structurally separate from the motor projection systems. It has special cortical areas of representation and these are connected by fibre systems with the sensorimotor executive apparatus on which tonectic excitations exert their influence. Regarding the frontal cortex from the central fissure to the anterior pole of the brain as subdivided into a prefrontal, a midfrontal, and a postfrontal (motor) region, the cortical tonectic zone is chiefly midfrontal. The striatum is an association or integration region where are regrouped the excitations from the differentiated cortical centers for tone. Wilson holds that higher (cerebral) centers concerned with tone must be conceded and that there is increasing evidence that muscular tone depends to some extent on the reaction between the „basal ganglia“ and the cortex and especially the intermedio-precentral cortex.

The midfrontal or intermedio-precentral region is the cortical seat or rendezvous of tone, the place to which stream the affects or tone impulses of the sensory centers of the cortex. Separate cortical tonectic centers constitute together a cortical tonectic zone. This area is not necessarily topographically entirely segregated from the pyramidal motor cortex. Each motor center probably has its separate outlying cellular tonectic annex or dependency, these centers of tone lying in the main anterior to the motor centers proper. As most of the pyramidal motor system is represented in the postfrontal region, chiefly in the precentral convolution, the tonectic centers are mainly located anterior to this convolution. Scattered over the midfrontal region however are some pyramidal motor centers, as I have demonstrated with Dr. Charles H. Frazier by faradization of the midfrontal and prefrontal region of the human brain. Here movements of the jaw have a wide representation, and here in a region several inches cephalad of the central fissure are centers concerned with movements of Darwin's muscles of grief, movements of the orbiculars of the eyelids, the pyramidals of the nose, the corrugators, and the inner fascia of the frontalis. Somewhat in advance of the precentral convolution are also represented such movements as turning of the head to one side, forward bending of the head, and closure of both eyelids. Nevertheless, the major portion of the midfrontal cortex is ~~non~~ not excitable by the electrical current as are pyramidal motor centers. The ~~non~~excitable centers are concerned with the maintenance of tone.

In a paper on the cerebral mechanism of emotional expression[1] I expressed the view that in the midfrontal region of the cerebral hemispheres, especially on the right side in right-handed persons, is an area which may be regarded either as an independent zone or as an anterior subdivision of the general motor zone, holding that here are cortically represented those movements which are especially concerned with the expression of emotion, the term movement in this connection being applied not only to the movements performed by skeletal muscles, but to movements visceral, vascular, secretory, etc., in fact all which result in any way in adding to the sum total of emotional expression. Rire spasmodique and pleurer spasmodique, however, are after all in large part phenomena of abnormal tonicity, so that involuntary or spasmodic emotional expression belongs to the general subject of abnormal muscle tone. The cortical zone of representation, therefore, includes both the precentral cortical tonectic and the cortical motor centers especially concerned with emotional expression.

---

[1] Transactions of the College of Physicians of Philadelphia, 1912.

If the midfrontal region is especially concerned with tonicity, cases of disease supporting this view should be forthcoming. Reports of such cases are not numerous, but are not entirely lacking. In the case of Massalongo[1] which in the rapid development of its motor symptomatology passed through the entire gamut of abnormal movements including fascicular tremor, athetoid and choreiform movements, tonic and clonic spasms and convulsions, the lesions present were a well defined tuberculous meningitis affecting especially the frontal lobes of both hemispheres. The motor symptomatology in this case was more complex than that which is present in a case limited to the pyramidal cortex or subcortex. In a case of SCHAFFER[2] cited by MINGAZZINI, the recorder attributed spasmodic laughter to the softening and atrophy of the right frontal lobe. In a case of my own in which operation revealed extensive destructive disease of the right midfrontal and prefrontal region, one of the manifestations was a smiling or laughing aura which was sometimes followed by left facial spasm, then by convulsive movements on the left or on both sides of the body. Many years ago I recorded a case of tumor of the brain involving the first and second frontal convolutions, the gyrus fornicatus and slightly the callosum.[3] Besides the general symptoms of brain tumor the patient exhibited among other focal symptoms mental slowness, loss of attention, hysterical sobbing, nystagmus, tonic spasm of neck and forearm, some hypertonicity of both legs, volcanic outbursts of speech, polyphagia, and oscillations both of body and head temperature. Petrina reported a case of bilateral tumor or tumors almost destroying both frontal lobes.[4] Among the symptoms were some weakness of mind, emotionalism, choreic movements of right arm increased by mental excitement, erotic conduct, tremor in both arms, automatic movements in right arm and contractures at both elbows. WILSON's cases of perseveration[5] associated with tumor of the midfrontal region must be borne in mind in considering this phase of the subject. A thorough study of the literature would probably reveal a considerable number of cases illustrating the points brought out by the few references here made.

A consideration of all the published data at command regarding the connections of the frontal cortex with the striatum indicates that the weight of opinion is in favor of the nonexistence of a projection system from this portion of the cortex to the striatum, but that the two regions are closely connected by association fibres. The projection fibres which have been shown to pass from the frontal cortex to the striatum (BIANCHI and ALGERI, MARINESCO and others) probably pass through the caudatum and pallidum on their way to the thalamus, subthalamus or ruber. The projection tract demonstrated by von Monakow and Archambault through their studies of rubral degeneration in destructive frontal disease also may take this course.

---

[1] Revue Neurologique, 1903.

[2] Stuttgart 1904.

[3] Medical Times. Jan. 18, 1879.

[4] Vierteljahrschr. f. d. prakt. Heilk. Bd. I. 1877.

[5] Transactions of the Section on Neuropathology. The Seventeenth International Congress of Medicine. 1913. p. 106.

It is generally accepted that morphologically the striatum is a part of the cortex. It is probable that the best solution of the problem of the functions of the striatum, both caudatum and lenticula, is to be found in the assumption that physiologically as well as morphologically this basal ganglion, so-called, functions like a region of the cortex. Just as primary and secondary cortical visual and auditory centers and areas are recognized, and in the same sense that the fronto-insular speech center may be regarded as a special cortical association center, so the striate nuclei may be looked upon as cortical regions closely related in function with the midfrontal or midfrontal and a part of the prefrontal cortex. Regarding the latter as a region where differentiated tonectic centers have their seat, the striatum is probably a cortical area in which these differentiated centers are integrated. According to Ganser (cited by Brodmann) complete removal of a hemisphere to such an extent that the striatum lies exposed produces no noteworthy atrophy in the latter, this behaving in some manner as a region of the cortex whose neighbouring parts have been destroyed, and suffering only through the involvement of association fibres.

When the differentiated cortical tonectic centers auxiliary to the motor cortical centers are released by destructive lesions from their striate connections, their tone stimuli probably overflow by gyral associating tracts into contiguous and correlated motor centers. Such overflow necessarily produces an arhythmia of movement because of irregular and unchecked stimulation.

It is necessary to recognize two motor projection systems in order to explain the phenomena of movement in their relations to cortical activity. The first of these is the pyramidal system which needs only to be mentioned. A second projection system must be postulated for the nonstriated or smooth muscle system in so far as this is activated by cortical processes which result in the combination of skeletal and non-skeletal movements. The influence of emotions like fear, rage, and excessive joy on skeletal movements associated wich vascular, pilatory, glandular, and visceral motor functions can only thus be explained.

„The parts of the organism“, says Cannon, „which are peculiarly activated in affective states are the glands and smooth muscle of the viscera. Although hurried breathing, and trembling, and characteristic bodily postures and facial expressions implicate the skeletal muscles, these muscles, under the will, may be sent into similar patterns without emotional experience, or in the throes of an emotional disturbance they may be set in deceptive patterns of quite opposite meaning, for example, we can smile through our tears. The pouring out of tears, however, and the 'cold sweat', the dilation of the pupils, the erection of hairs, the pallor from contracted blood-vessels, the rapid heart-all these are changes from the routine of life, in organs not subject to voluntary control, in organs which are only roused thus from their normal even tenor by the rush of a wave of feeling. So characteristic are these disturbances of the viscera in affective states that they have been regarded in the well known **James-Lange** theory, as giving rise to the peculiar qualities which differentiate emotional experiences from one another."[1]

The cortical tonectic centers related to these emotional movements are situated more anteriorly than those for skeletal movement in general. They probably act through a fronto-rubral projection tract on the rubro-spinal system. Through the deficit or increase of activity are to be explained the vasomotor, secretory and other symptoms which are present in both fronto-cortical and caudate lesions. Excitations from the frontal cortex reintegrated in the caudatum and lenticula pass to the thalamus and subthalamus and thence by thalamo-cortical pathways to the motor centers of both the pyramidal motor and the fronto-rubral systems.

The observations of **Wilson** show a direct connection by way of the ansa lenticularis with the ruber. My own case seems to indicate a similar connection and in addition one between the caudatum and the ruber. Not improbably there is a reflex arc by way of this thalamo-strio-rubral path. Spontaneous laughter and weeping in young children and in adults not under the influence of strong feeling may perhaps be explained by this supposition, whereas when the will, or strong emotion entersinto emotional expression the frontal cortex comes into play.

In the facts presented in the study of the case here recorded in detail are some indications, at least, of a caudate syndrome as distinguished from that resulting from lesions confined to the lenticula. Very briefly stated this symptom complex included hypertonicity and tonectic paresis as exhibited especially in the phenomena of involuntary painful emotional expression and various symptoms referable to the cortico-autonomic nervous system, vaso-motor and secretory affections, disturbances of temperature, pulse, respiration and of various forms of glandular activity. It will be recalled that the necropsy and microscopical examination showed that the right caudatum and the anterior limb of the right internal capsule had disappeared, while the head of the left caudatum, although much atrophied, did not show more than one-half the destruction of its fellow on the right, and the anterior limb of the left internal capsule at higher levels was intact. Both the subthalamus and ruber were about one-third smaller on the right than on the left. This greater degeneration of the right subthalamus and ruber may have been related to the complete destruction of the right caudatum and anterior capsule.

The literature of the subject under consideration is very large although much of it is unsatisfactory. It includes the extensive literature of involuntary or spasmodic emotional expression in which appear the names of such well known neurologists as von **Bechterew**, **Nothnagel**, **Brissaud**, **Bianchi**, **Mingazzini**, **Constantini**, **Goldstein** and many others. Here are also to be found contributions of those who have written on the anatomy, physiology and clinicopathology of the striatum and subcortical ganglia in general, and on such symptoms as organic tremor, athetosis and chorea, and on all disorders characterized by changes in muscle tonicity. Prominent in this list are such names as von **Monakow**, **Dejerine**, **Obersteiner**, **Lewandowsky**, **Brodmann**, **Oppenheim**, **Anton**, **Gowers**, **Homen**, **Ormerod**, **Hughlings Jackson**, **Gordon Holmes**, **Vogt**, **van Gehuchten**, **Weir Mitchell**, **Mingazzini**, **Ross**, **Massalongo**, **Crocq** and many others. A complete bibliography would take many pages of the Neurologisches Centralblatt. Such bibligraphies are appended to the paper of **Gordon Holmes**[1] and to that of **Kinnier Wilson**[2] and are included in the well known works of v. **Monakow** and in the treatise of **Mingazzini**.[3] The term „epoch making" is sometimes used too readily, but it has a real significance in the case of the remarkable paper of **Wilson** on bilateral lenticular degeneration, etc., one of the greatest contributions to neurology in recent years. This paper has clarified our views with reference to the functions and diseases of the striatum in a way not previously approached.

---

[1] The American Journal of Psychology. Vol. XXV. Nr. 2. April, 1904.

For valuable assistance in the preparation of this paper I am indebted to my colleague Dr. T. H. **Weisenburg**.

---

[1] Brain. XXVII. 1904.

[2] Brain. XXXIV. 1912. Part 4.

[3] Anatomia Clinica dei Centri Nervosi. Torino 1913.

Druck von Metzger & Wittig in Leipzig. 969

SPONTANEOUS SUBARACHNOID HEMORRHAGE

A CONTRIBUTION TO THE SUBJECT OF MENINGEAL HEMORRHAGE*

SAMUEL LEOPOLD, M.D.
Instructor in Neurology and Neuropathology, University of Pennsylvania

PHILADELPHIA

Aside from the rare pachymeningitis hemorrhagica interna, and the traumatic pial hemorrhage, there has been no recognition of a spontaneous subarachnoid hemorrhage. Although this symptom-complex has been elaborately studied by Froin,[1] Chauffard,[2] Vigneras[3] and others among the French writers, little or nothing is mentioned of the subject by the American, English or German writers, Starr, Dana, Mills, Oppenheim, Finkelburg in Lewandowsky's[4] handbook, Schultze in Nothnagel's "System," and others.

In describing meningeal hemorrhage, Gowers does not differentiate the subarachnoid from the other forms, but his description corresponds very closely to this type. He says:

> Blood may be extravasated, (1) outside the dura, separating it from the bone; (2) beneath the dura; (3) beneath the arachnoid between it and the pia (subarachnoid hemorrhage). . . . Meningeal hemorrhage occurs apart from visible aneurysm under the same conditions as hemorrhage elsewhere in the brain. . . . In some cases in young adults, the occurrence of meningeal hemorrhage is probably indicated by intense pain in the head at one spot, soon followed by coma and convulsions. The cases are remarkable as occurring without other exciting cause than exertion in a hot room, or sometimes after a meal.

---

* Read before the Section on Nervous and Mental Diseases at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

[1] Froin: Thèse de Paris, 1904. Froin and Boidin: Gaz. d hôp. 1904, p. 9.

[2] Chauffard and Boidin: Presse Méd., 1903.

[3] Vigneras: Thèse de Paris, 1908.

[4] Lewandowsky: Handbuch de Neurologie.

Froin, in 1903, collected some reports of cases of subarachnoid hemorrhage but he included the traumatic form among them. Ehrenberg[5] was the first to separate the spontaneous subarachnoid hemorrhage from the rest. He reports two of his own, and analyzes twenty-seven other cases. He distinguishes two varieties, the one in which the meningitic picture predominates; the other an apoplectic onset, with coma or cerebral type. Since then Babinski[6] and Jumentie report two cases, and Forsheim[7] one.

Two of my cases illustrate the apoplectic form, and one the meningitic.

Case 1.—B. Y., white, aged 59, married, was admitted to the hospital April 25, 1910. Present illness began suddenly with vomiting, headache, pain in the epigastrium and stupor. (I had no opportunity of studying this case clinically and the notes taken are very meager.) The pupils were equal, reacted to light and accommodation. There were no paralyses. The patient died the next day. No clinical diagnosis was made.

At necropsy, examination of the brain showed a hemorrhage beneath the arachnoid at the base. It extended forward to the frontal lobes, and surrounded the optic chiasm. It covered the under surface of both the pons and the medulla. It covered the inferior surfaces of both hemispheres of the cerebellum. The convexity of the brain was free from hemorrhage. There was no evidence of meningitis or thrombosis. The skull showed no sign of fracture. Sections were taken from the pons and medulla for microscopic examination. Hemalum-fuchsin and Weigert stains were used. Sections of the cerebellum and the pons stained with hemalum-fuchsin showed a considerable collection of red blood-cells beneath the pia-arachnoid. The pia showed a considerable round-cell infiltration of the lymphocytic type. The blood-vessels showed periarteritis and endarteritis. The infiltration of cells around the vessels was intense in places. In two of the vessels in the pons the lumen was almost occluded. In the medulla, similar pathologic changes were noted. Sections of the pons and medulla stained with the Weigert showed a slight border-zone degeneration.

The pathologic picture was that of a syphilitic meningitis. Clinically, this case is interesting despite the meager notes, in that the onset is characteristic of this form of hemorrhage, and shows the apoplectiform type of seizure. The meningitic origin of the hemorrhage was only discovered on microscopic examination. As far as I can learn, no microscopic study of this condition has ever been reported.

---

5. Ehrenberg: Hygeia, 1912, p. 849.
6. Babinski: Soc. méd. d. hôp. d. Paris, 1912.
7. Forsheim: Deutsch. Ztschr. f. Nervenh., 1913, xlix.

The second case also illustrates the apoplectic form with coma.

Case 2.—H. M., an unmarried colored laundress, aged 19, with negative family history and previous medical history, two months ago had pelvic trouble, said to have been inflammation of the tubes. Patient had been well until the morning of her admission to the hospital, when she came home, complaining of severe headache, with vomiting. She went to her room, where she was found unconscious several hours later. She was admitted to the hospital that evening, Dec. 13, 1913, where she died several hours later.

Physical examination showed a young, well-developed girl. The pupils were unequal. There was rigidity of the neck, and spasticity of the lower limbs. Both patellar tendon-reflexes were increased. Babinski was present on both sides. The upper limbs were flaccid. The respirations were of the Cheyne-Stokes' type. Temperature, 100.4 F. The urine analysis showed no albumin, sugar or acetone. Microscopic examination, negative. The diagnosis of subarachnoid hemorrhage was made, and lumbar puncture advised. The possibility of a meningitis was also considered. Hysteria was easily excluded. Uremia and diabetic coma were excluded by the urinary findings. There was no history or visible sign of trauma. Lumbar puncture was made, and the fluid came out drop by drop, somewhat rapidly, until twenty c.c. were collected in two tubes, and both showed a diffuse red color, considerably lighter than pure blood. On standing, no clot was to be noted in either tube, only a slight granular sediment. Centrifuging the fluid showed the supernatant fluid of a yellow color. The Wassermann reaction was negative. No relief followed the withdrawal of the fluid. One hour later the patient died. No necropsy was permitted.

Summary.—A young woman, previously healthy, suddenly was seized with headache, vomiting and unconsciousness. The symptoms present were rigidity of the neck, and spastic paralysis of the lower limbs with Babinski. Clinical diagnosis, spontaneous subarachnoid hemorrhage.

Case 3.—Italian stone-worker, aged 23, family history and previous medical history negative, one month ago complained of fever, backache, headache and general weakness. He was treated in an Italian hospital for two weeks, and was apparently cured, though still weak. Five days after his discharge he suddenly developed severe headache, pain in the back of the neck and persistent vomiting, and was admitted to the hospital, complaining of his head and pain in the back of the neck.

Examination showed a young man of fair nutrition, somewhat apathetic. When questioned, patient pointed to his head. Pupils were equal, reacted to light and accommodation. There was rigidity of the neck. The limbs showed no paralysis.

Kernig's sign was present. The knee-jerks were absent. There was no Babinski or ankle-clonus. The eye report showed deviation of the eyes to the right, though at times they moved to the median line. The media were clear, the disk margins slightly hazy, but the vessels were neither engorged nor tortuous. Ear examination was negative. Temperature 100 to 101.4. The lungs showed slight impairment. The diagnosis was subarachnoid hemorrhage with tuberculous diagnosis was subarachnoid hemorrhage with tuberculous meningitis. Lumbar puncture was ordered.

At necropsy, the viscera showed a general miliary tuberculosis. The brain showed miliary tubercles scattered over the convexity and at the base along the vessels. Besides a subarachnoid hemorrhage at the base extended from the optic chiasm over the peduncles, slightly involving the temporal lobes on each side, and also over the pons and cerebellum. The hemorrhage was most marked in the right cerebellopontile angle. There was some hemorrhage over the vermis. The convexity of the cerebral hemispheres was free from hemorrhage.

Summary.—Young man. History of general weakness for a month. Five days before admission to the hospital was seized with headache, vomiting and meningitic symptoms. Examination showed rigidity of the neck, Kernig's sign, loss of knee-jerks, no Babinski, no paralysis of the limbs. Clinical diagnosis; tuberculous meningitis, with subarachnoid hemorrhage. Pathologic Summary: General miliary tuberculosis of lungs, liver, kidney and brain, subarachnoid hemorrhage.

There is some doubt whether this case should be classified among the pure forms of subarachnoid hemorrhage. The picture here is rather that of a secondary complication in the course of a miliary tuberculosis.

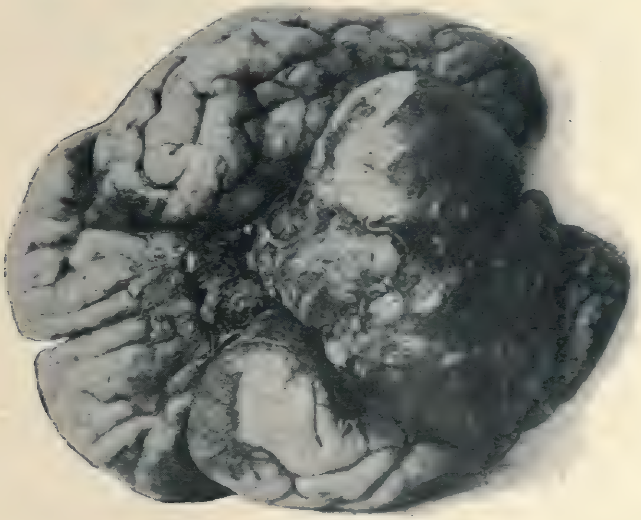
The sudden onset in these cases with meningeal symptoms, with or without coma, the disproportion between the onset, severity of the symptoms, and the range of temperature, the presence of hemorrhage in the cerbrospinal fluid, will usually differentiate this condition from other cerebral states. It may not always be possible to prove a primary meningitis as the underlying factor. The etiology has been variously ascribed to sunstroke, as in Bouquet's[8] case, in which the patient, following prolonged exposure to the sun, suddenly developed rigidity of the neck, headache and Kernig's sign. Lumbar puncture showed hemorrhage. Relief of symptoms followed immediately. Disturbed or aberrant menstrual function was the cause, according to Follet and Chevrel[9] and Gaillard and Boyé,[10] in their cases.

---

[8] Bouquet: Ref. Rev. Neurol., 1909.

Follet and Chevrel's patient, a domestic, aged 21, was suddenly seized during the menstrual period with weakness, and fell to the floor unconscious. She awoke with intense headache and had persistent vomiting with symptoms of meningeal irritation. Lumbar puncture showed hemorrhage.

In Gaillard and Boye's case, there was no evidence of trauma, syphilis, exposure, emotion or infection. The interesting fact was noted that an amenorrhea following oophorectomy had existed for one year.



Area of hemorrhage in Case 1.

Violent emotion is attributed as the cause in one of Froin's cases, overexertion in Forsheim's. Syphilis was noted in seven of the cases reported. Tuberculosis was present in one of Froin's cases, and in my Case 3. Chronic nephritis and hemophilia have also been reported as causes. It is possible that meningitis will prove to be the underlying cause in the majority of cases. Babinski believes that syphilitic meningitis plays a distinct rôle in these cases.

---

[9] Follet and Chevrel: Gaz. d. hop., 1910, p. 547.

[10] Gaillard and Boyé: Bull. Soc. méd. d. hôp. d. Paris, 1909, p. 1268.

The localization of the hemorrhage at the base, so characteristic of this form of hemorrhage, would add further support to the meningitic origin.

While the symptomatology in the cases I report and those found in the literature shows the ordinary signs of meningeal irritations and coma, several features impress themselves. Headache was localized either over the occiput, or at the base of the neck. Sometimes the pain radiated down the spine and in one of my cases was localized over the epigastric region. Rigidity of the neck and Kernig's sign were present in two of my cases, and one or other of these symptoms was present in all cases reported. This meningitic picture is most favorable for recovery.

Coma may be the initial symptom, followed either by death or the appearance of meningeal symptoms. In a few cases a condition of recurring coma was noted. All the fatal cases showed coma.

Jacksonian convulsions were noted by Froin and Babinski. In these cases the hemorrhage had escaped to the cortex and clots were found over the motor area. The reflexes were increased in some cases and absent in others. Babinski's sign was also inconstant. The paralytic symptoms were manifold, according to the extent and localization of the hemorrhage.

Ocular palsies, conjugate deviations of the eyes, paralysis of one or more limbs, transient hemianopsia, all have been noted.

The value of lumbar puncture in these cases is apparent. In all suspected or doubtful cases in which lumbar puncture was performed, the presence of blood without clot was obtained. Many of these cases were confirmed by necropsy. In fact, according to Follet and Chevrel and others, no diagnosis can be made without lumbar puncture.

The accidental puncture of a vessel during the procedure may be distinguished, according to Milliet, from subarachnoid hemorrhage by collecting the cerebrospinal fluid in three tubes. The former will show streaks of blood in the clear fluid, also the presence of clot, while the latter is a diffuse red throughout, and shows no clot. Centrifuging the fluid will also show the supernatant liquid of a yellow or slightly reddish tinge. According to Widal,[11] this is the only pathognomonic sign of hemorrhage. In only one other condition, that of jaundice, is this sign to be found.

Lumbar puncture has proved of therapeutic value in some of the cases, relief following one or more punctures.

348 South Sixteenth Street.

---

[11] Widal: Presse méd., 1903.

Reprinted from The Journal of the American Medical Association
Oct. 17, 1914, Vol. LXIII, pp. 1362-1364

Copyright, 1914
American Medical Association, 535 N. Dearborn St., Chicago

PROGRESSIVE LENTICULAR DEGENERATION

WILLIAMS B. CADWALADER, M.D.
Instructor in Neurology and Neuropathology at the University of Pennsylvania, Department of Medicine; Clinical Assistant at the Orthopaedic Hospital and Infirmary for Nervous Diseases
PHILADELPHIA

Although progressive lenticular degeneration, first described under that title by S. A. K. Wilson[1], is well established clinically, it has not often been encountered in this country. Since Wilson's first publication, in 1912, a number of cases have been recorded in foreign literature, but, with the exception of a case recently reported by Auer[2] and some brief references made before the neurological societies of New York and Philadelphia, little or nothing on the subject has appeared in American literature.

The patients whose histories are recorded here were admitted at different times to the wards of the Orthopaedic Hospital and Infirmary for Nervous Diseases, Philadelphia, and first came under the care of Dr. S. Weir Mitchell, and later passed to the service of Dr. John K. Mitchell. They have both been under observation for a considerable length of time. Examinations have been made repeatedly by various observers, and all the facts have been corroborated carefully from time to time.

CASE 1.—*History.*—B. R. H., man, aged 18. Patient's parents are healthy; a grandaunt was believed to have had myxedema; a first cousin and also an aunt had exophthalmic goiter. The patient was born at full term, the labor having been normal. He first walked and spoke at the usual age, and developed normally. He has never had convulsions or been otherwise ill. He was considered an unusually intelligent and attractive boy, always at the head of his class at school, and but for illness would have entered college at the early age of 14.

---

[1] Wilson, S. A. K.: Brain, March, 1912.
[2] Auer: Am. Jour. Nerv. and Ment. Dis., June, 1914.

Present condition began when the patient was 14 years of age, while he was at boarding-school. The symptoms developed rather quickly, first manifesting themselves in the form of sluggish movements, indifferent table manners, a constant driveling of saliva, dysarthria and general lassitude. The condition continued for a number of weeks, and then the symptoms became more and more exaggerated. When first seen by his physician about six weeks after the onset, the patient appeared to be a driveling idiot, exhibiting general thickening of the skin and peculiar jerking movements of the extremities. A diagnosis of myxedema with multiple myoclonus was made, and thyroid extract administered. The mental improvement that followed was said to have been remarkable and almost immediate. Other symptoms attributed to the myxedema disappeared gradually in from one to three months, but the tremor became more pronounced. The thyroid medication was continued until the patient was 18 years of age. During the past two and a half years the patient's condition has remained stationary.

Physical Examination.—The facial expression is often quite vacant, especially striking being the more or less constant smile he wears. His complexion is dark and his hair is black; the skin is not thickened and in no way does it suggest a myxedematous change. Patient states that his complexion was always dark.

The pupils are equal in size and react normally to light, accommodation and convergence. The optic disks are normal, and the ocular movements are performed regularly; there is no nystagmus. When the patient winks, the eyelids occasionally remain closed and the lids cannot be elevated voluntarily without assistance. This appears to be due to a tonic spasm or contracture of the muscles of the eyelid. The mouth is not held open continually, but gapes more often than in most normal persons. The flow of saliva is not increased. The movements of the lower jaw, the tongue and the muscles of the pharynx and larynx are all performed with normal power. Laryngoscopic examination showed that the movements of the vocal cords were quite good, but somewhat slow. The hearing is normal. There is no difficulty in swallowing either liquids or solids. The voice is high pitched and has a nasal quality, and dysarthria is pronounced, being characterized by the slurring of syllables and the cutting off of the ends of words; the speech is quite different, however, from that of multiple sclerosis, in that the scanning tendency is lacking.

The movements of the facial muscles suggest a condition of spasticity. When tested by the customary methods, each of the cranial nerves appears to be normal. The thyroid gland is not enlarged, and the superficial lymph-glands likewise present no evidences of enlargement.

The left sternomastoid muscle is in a state of extreme hypertonicity, in consequence of which the head is often turned to the right; this is not, however, constant. The head can be rotated freely, but the rotation is accompanied by a fine tremor.

The musculature of the entire body and extremities appears to be good, but there is little subcutaneous fat, so that the outline of each muscle is well defined; nevertheless the patient does not appear to be emaciated. The skin everywhere is of a copperish-yellow hue, and a few small pigmented moles are scattered over the body. The most striking features are marked tremors, rigidity and a tendency to contractures, all of which are never absent, although they vary in intensity.

The upper extremities are in a state of almost continuous clonic tremor, more marked distally than proximally, and somewhat more exaggerated on the right side. The excursion of movements is wide, and the rate is rapid, but variable. On the slightest excitement or on exertion the tremor becomes violent. On attempting to place the palm of one hand against the cheek the hand strikes the face forcibly in a rapid succession of rhythmic flexor and extensor movements. A similar tremor of the lower extremities occurs while the patient lies in bed and on attempting voluntary movement of the trunk; it is also present when he is in the sitting and the standing positions. On starting to walk and in turning, one foot, generally the right, is thrown into a series of rapid clonic movements, the foot tapping the floor violently. When an effort, however, slight, is made to restrain the muscular movements, the tremor shows a decided tendency to involve the musculature of the entire body. At times, when the patient is standing quietly with his hands hanging at his sides, or when weights are placed in his hands, the tremor may disappear almost entirely for a few moments. The muscles of the entire body are in a state of more or less hypertonicity. The left foot is frequently in a condition of extensor spasm, assuming the equinovarus position, with the large toe flexed dorsally. On standing or in starting to walk there is often overextension of the trunk, and consequently a tendency to fall backward; equilibrium can be regained, however, by muscular effort. In this it differs from the uncertainty of ataxia, and appears to be due to muscular rigidity.

The abdominal muscles are tense and fixed, and the abdominal reflexes are equal and otherwise normal. Voluntary movements, such as are made in walking, are "wooden" and "mechanical" in character, and lack the smoothness that characterizes the working of a well-oiled machine, to which the normal body movements may be likened. Because of rigidity all voluntary movements are greatly retarded, yet the patient can perform flexion and extension of all joints, as well as adduction and abduction. Walking and rising from the sitting and prone position can be accomplished without assistance, but not, however, without considerable voluntary effort on the patient's part. The muscular power is unimpaired and does not differ in the extremities of the two sides, as estimated from the resistance exerted by the examiner. There is no paralysis present. With the exception of the incomplete contractures of the peroneal muscles of the left leg, the left sternomastoid and the elevators of the eyelids, previously mentioned, actual permanent contractures are absent.

Pain is never present, nor can any sensory change be discovered. The sensations, as tested for touch, heat, cold and pain, are everywhere normal.

The tendon and the cremasteric reflexes are all equal and entirely normal; they are active, but not exaggerated. There is no ankle-clonus, and no Babinski reflex is present. Owing to rigidity, overextension of the great toe sometimes occurs, and this could be mistaken for the Babinski sign.

Examination of the thoracic and abdominal organs reveals no abnormalities; the liver dulness is not diminished and appears to be normal. The systolic blood-pressure is 118. The electric examination gave entirely normal reactions. The blood and urinary examinations were negative, and there was no evidence of bile-pigment or other abnormalities in the urine.

The mental condition of the patient was fairly good; he was intelligent, although he lacked the power to concentrate, and was distinctly foolish and inclined to be childish, but apart from this nothing characteristic could be discovered.

CASE 2.—*History.*—W. P., man, aged 36. Father is healthy; mother has had intermittent attacks of melancholia; one brother is well; one older sister and two brothers are afflicted with a condition similar to that presented by this patient, namely, tremors, spasticity and contractures of the muscles, which began in youth. This statement has been corroborated, but personal examination was not made. One of these brothers is mentally deranged, and displays outbursts of temper and other manifestations of disordered mentality. The other brother and sister are normal mentally.

This patient, as well as the other affected members of his family, was born at full term and developed normally. The present condition began in 1892, when the patient was 16 years old. The tremor at first affected the right side, the muscular rigidity and spasticity gradually increasing and spreading to the muscles of the neck, causing tremor of the head, and later involving the entire body and all the extremities. The patient was first seen in 1900, and has remained under observation ever since. He now presents a condition characterized by constant involuntary movements, the tremor increasing with emotional disturbances and voluntary effort, but not disappearing when he is at rest. The tremor is fairly regular, of the type which manifests itself by flexions and contractions, involving almost every group of muscles in the body; dysarthria is pronounced, but his speech is not marked by scanning.

Spasticity and rigidity of the muscles, as well as contractures are pronounced. The contractures are not permanent, however, except that affecting the larger muscles of the lumbar region, which causes the trunk to be bent to one side.

His physical inability seems to be due entirely to tremor, rigidity and apparent incoordination, and not to paralysis. All the tendon reflexes are slightly exaggerated, but equal. The skin-reflexes are normal. The Babinski sign is absent. Mentality is and always has been normal.

The disease was progressive at first, but five or six years after its onset it became stationary. Further examination shows nothing abnormal. The eyes present no abnormalities; there is no nystagmus, and sensation is normal. Both Wassermann and Noguchi reactions are negative. All thoracic and abdominal organs are normal, and there are no parasthesias; the sphincters functionate properly, and there is no ataxia or pain. The liver dulness is not diminished, and the patient asserts that he feels well. Electric examinations gave negative findings, and the urinary examination was also negative.

The clinical picture presented by these two patients differed but slightly, this difference being more particularly one of degree than of character. The two cases appeared to be of the same type. In Case 2 the disease seems to have commenced gradually, assuming a chronic nature from the beginning, but evincing no constitutional symptoms, the tremor having been the first visible evidence of the disease. This onset is similar to the one described by Wilson as chronic. In Case 1 the onset seems to have been acute, and the symptoms were regarded as myxedematous; after some weeks, however, these symptoms all subsided, but the tremor, rigidity and tendency to muscular contracture remained. The patient's mentality was impaired, and he displayed a tendency toward childishness. From the family history, and owing to the fact that the acute symptoms disappeared while thyroid extract was being administered, it might be assumed that some disturbance of the ductless glands was the etiologic factor; whether the acute symptoms merely subsided, as they always do in the beginning, or whether they really disappeared as the result of the thyroid treatment, is, indeed, questionable.

In neither case was there any clinical evidence of the presence of hepatic disease. The liver dulness was neither diminished nor increased in either case. The urine of both patients showed no abnormal characteristics, bile-pigment and the like being absent. The first patient did, however, exhibit a peculiar yellowish color of the skin, different from that of jaundice. This discoloration is undoubtedly similar to that described by Fleischer[3] and Rumpell.[4] Volsch's[5] (1911) patient also showed pigmentation of the cornea, a finding that was absent in my case.

The significance of corneal pigmentations when associated with tremor is still very uncertain, particularly when these are accompanied by skin changes and evidences of hepatic disease. This subject has recently been carefully studied by Holloway.[6] The first patient presented a very curious condition, in that he was unable to open his eyelids without assistance. In referring to this in his history it was spoken of as a form of contracture. I have never observed anything quite like it, and if it is part of the picture of lenticular degeneration, this manifestation has not previously been reported. At times when both eyes would be closed naturally the eyelids would remain closed and could not be opened voluntarily; when, however, the patient placed his index-finger on one orbital ridge and pushed upward, both lids could be elevated and the eyes remained open. This condition was markedly aggravated by any emotional disturbance, although the latter did not appear to produce the condition.

A relationship seems to exist between the disease known as progressive lenticular degeneration and the condition named by Westphal pseudosclerosis, the two diseases having some points of similarity. In December, 1912, I presented the patient here described as Case 2 before the Philadelphia Neurological Society as a case of pseudosclerosis, and called attention at the time to the possibility of the existence of lenticular disease. Since then Strumpell[7] has reported a similar case, drawing attention to the similarity or relationship of these two curious clinical pictures. Westphal[8] himself has reported a case of pseudosclerosis with necropsy findings. He was able to discover distinct changes in the nervous system, chiefly of the glia nuclei, and more especially in the gray substance of the large ganglia of the cerebrum, and also in the dentate nucleus of the cerebellum. The nuclei of the ganglia-cells were enlarged, altered in shape, and were deficient in chromatin substance. In addition, the liver was distinctly reduced in size and presented a cirrhotic appearance. In reporting this case Westphal calls particular attention to the relationship that pseudosclerosis seems to bear to this peculiar morbid process, characterized by pigmentation of the skin, cirrhosis of the liver, tremors and mental and nervous phenomena. It is my belief that the first case here recorded might belong to this group.

---

[3] Fleischer: Deutsch. Ztschr. f. Nervenh., 1912, xliv, 179.
[4] Rumpell: Deutsch. Ztschr. f. Nervenh., 1913, xlix, 54.
[5] Volsch: Deutsch. Ztschr. f. Nervenh., 1911, xlii, 335.
[6] Holloway: Am. Jour. Med. Sc., August, 1914, p. 235.
[7] Strumpell: Deutsch. Arch. f. Nervenh., 1914, l, 455.

1710 Locust Street.

---

[8] Westphal: Arch. f. Psychiat. u. Nervenkr., 1913, li, 1.

Reprinted from The Journal of the American Medical Association
Oct. 17, 1914, Vol. LXIII, pp. 1380-1382

Copyright, 1914
American Medical Association, 535 N. Dearborn St., Chicago

PSYCHOSES FOLLOWING APOPLEXIES.

By JOHN H. W. RHEIN, M. D.,

Professor of Diseases of the Mind and Nervous System at the Philadelphia Polyclinic and College for Graduates in Medicine, Neurologist to the Howard Hospital, Physician to the Philadelphia Home for Incurables, Philadelphia, etc.

A review of the literature will reveal the fact that post-apoplectic psychoses have received less consideration than the subject deserves, in fact not a great deal has been written about this symptom-complex.

That a perverted mental state occurs after an apoplexy in a certain number of cases has always been recognized, but the text-books on the whole have described this condition in a more or less perfunctory manner.

The usual and best recognized form of mental change after an apoplexy consists of a simple moderate intellectual defect characterized by failing memory, emotionalism and defective will. Beside this, however, there may develop a progressive dementia, best described as dementia post-apoplectica.

But more rarely there occurs a certain mental state which has features which are more or less characteristic of a post-apoplectic psychosis. It is to illustrate the last, that I venture to report two cases which recently have come under my observation in private practice and which I have had the opportunity of studying from the very onset of the mental symptoms, an event which is not often afforded the alienist who is more apt to be called to the care of such cases later in the course of the disease.

The following cases are practically identical in their clinical features and seem to present a phase of post-apoplectic psychoses which is infrequently observed.

Case i.—M. H., aged 54. Father died of pneumonia; mother died of apoplexy and showed some mental change before the attack. A maternal uncle died of apoplexy, and a maternal sister died in an institution for the insane. Two brothers were living and well. One brother died of tuberculosis and one brother in infancy.

The previous history was negative, except that for some years she has suffered from pelvic trouble, dependent upon an anti-flexed uterus. The menopause occurred at 49 years of age.

For two or three months before the attack of apoplexy she had complained of nervousness consisting of irritability, being readily disturbed upon slight provocation and of a tendency to fall asleep in the trolley cars. There was also sugar found in the urine on one occasion. Under appropriate diet and general measures the sugar disappeared and there was considerable improvement in her general condition.

On January 14, 1913, she fell to the ground without loss of consciousness. This occurred on two or three occasions during the ensuing two days, and finally on the 16th of January after falling again the left arm and leg and left side of the face became paretic. Consciousness was not disturbed and the mental condition was good. On the fourth day the patient became emotional and depressed, complaining of headache and the paralysis became more profound until at the end of ten days she was completely paralyzed in the left side of the face, in the lower distribution and in the left arm and leg.

About the end of the first week there developed restlessness, depression, complaint of pain in the head and affected limbs. Less than three weeks after the attack she became noisy by night and during the day she was very despondent, crying much of the time, shouting for help and complaining that those about her were not affording relief and were neglecting her.

Later she believed that many people were in the same bed with her, that she had two heads and that the bed was on fire. She became completely disoriented as to time and place.

These mental states at first occurred in attacks lasting one or two hours. In the interval she moaned with pain, or cried bitterly and was depressed.

The episodes varied in character. She was disoriented all of the time. She was always away from home, sometimes in a hospital attached to a church in an adjoining parrish; sometimes she was living in another world; sometimes she was in a convent and controlled by priests; sometimes she was confined in a house of ill-fame and was surrounded by half-dressed men and women from whom she could not escape. She had been taken to the house of ill-fame by a young fellow who drove her there after forcing her into a cab. This was a great disgrace and the day following this episode she thought she should go to communion.

Later these delusions persisted for many hours and days, changing from one to the other from time to time. They showed the following variations: She had gone to visit her parents (who were dead); liniments applied to her limbs were poison; the nurse put poison in her food and the medicines injected or given by mouth were poison. Paint was put in the milk which she refused for this reason. She wept bitterly over the funeral of a big fish. The Queen of Sheba had presented her with jewels. She was greatly depressed over the belief that she was gradually bcoming a negro. Her bed was a carriage and her feet and legs the horses. She thought she was dead and in a coffin, and talked a great deal about funerals and undertakers.

Her legs were fish and her hand a baby. Her leg had been crushed in an accident and had been amputated. She had two heads.

She manifested most of the time hallucinations which varied in character. Italians were in the house, and dogs wandered about her bed. Repeatedly large numbers of fish filled the air of her room, some walking, some flying. The faces of maimed children and babies were often seen.

She was very noisy especially at night. She resented the lateness of the visits of her physicians and when they moved her during examinations giving her pain, it was done on purpose to hurt her. She repeated words and phrases over and over again. (Perseveration.)

This condition persisted until March 30, 1913, two months after the attack when she underwent a febrile reaction lasting ten days in which the temperature rose to 102°, and was associated with vomiting and abdominal distention. After this attack she improved quite rapidly in all ways, until three weeks later she became quite normal mentally.

She ceased to complain of pain and there was also considerable return of power in the paralyzed limbs. She improved to the point of being able to take a few steps assisted.

An early and persistent symptom was incontinence of the urine and fæces. The examination of the heart and lungs was negative and the urine showed at times traces of albumin, and occasionally a few granular and hyaline casts. At no time could the kidneys be said to be a factor in the causation of the mental or other symptoms. The arm jerks and knee jerks were increased and more so on the left side and the Babinski phenomenon was present on the left. No change in sensation was noted, and there was no hemianopsia.

She stated after her recovery mentally that she thought that all the time she was sick she was in an opium den, or that she had been travelling or was among enemies. The hypodermics administered during her illness she believed was associated in her mind in some way with the morphine habit which she thought she had contracted. She remembers seeing horrible sights but the most painful memory was that she was confined in some house and could not get away to go home.

The mental condition in this case may be summarized as follows: There were maniacal outbursts; delusions mainly painful and persecutory; hallucinations, phantasmagoric and depressing; disorientation, and perseveration. Especially to be noted is the great complaint of pain in her head and palsied limbs, and the depressive state.

Case 2.—A. C., aged 64. Father died at 77 of senile change and heart disease; mother died at an early age of phthisis; one brother died of heart disease; otherwise the family history is negative.

Fifteen years before the attack of apoplexy she had had an attack of neurasthenia and thereafter began having stomach trouble which persisted until a year before her attack, when a rest cure was given and she recovered, remaining in good health until October 3, 1912, when she complained of continual pain in the right side of her head. On October 4, she awoke and found her left arm useless but she recovered from this in the afternoon except that she dropped things from her hand from time to time.

On October 7, 1912, she fell down from weakness in her left leg and felt weak in her left arm. At the same time the pain in her right temple became intense. On October 12, 1912, suddenly there was some confusion of speech and on the following day she lost power in the left arm and leg, the paralysis becoming complete in four days. From the beginning there was continual complaint of pain in the right temple and in the left arm and leg. She was very restless, moaning constantly and complaining that those about her would not give her relief. Two days later she showed some mental confusion. She believed she was on a boat, saw relatives in her room (who live miles away), thought she was in Atlantic City and saw soldiers and processions in her room. Then in a few days she saw phantoms and conversed with imaginary people. She was noisy and cried out at night and passed her urine involuntarily. She talked constantly, except when asleep, complaining of the unkindness of those about her, turning against her daughter her nurse and doctor and slept only under the influence of hypnotics.

She believed she had gone to New York on the 1.45 p. m. train and had come back at 6 p. m. the same day. She said she was not tired and stood the journey very well indeed, having walked around a good deal in New York and had gone to see a play called, "When Bunty Pulls the Strings."

She was most of the time very much disoriented as to time and place. She was very noisy especially at night, complaining a great deal of pain in the head and affected limbs and shrieking so loudly that the neighbors could hear. She was depressed most of the time abusing those about her for not giving her more relief. She often saw dogs and children's faces about her.

On December 2, 1912, there was some improvement in her mental condition for a short time but in a few days again she became irrational, very confused and talkative, moaning and crying. She was in a pulman car again, did not recognize her daughter, her doctor, or her nurse, talked continuously to imaginary people, and was still very noisy.

On January 4, 1913, she became stuporous, moaning and crying loudly, a condition which persisted until about March 22, when she began to clear up, mentally, recognizing everybody and conversing intelligently. Her mental condition continued very good for a month when, after an attack of the mumps, she again complained of great pain in the left leg and arm and cried most of the time although otherwise was clear in her mind.

The heart and lungs at no time showed any abnormality and the urinary analysis was negative. The reflexes were generally increased, more markedly so on the left side. No sensory changes were noted and an examination of the fields of vision failed to reveal any hemianopsia.

To summarize, the mental state was characterized by mild hallucinations; delusions somewhat persecutory and depressive; disorientation and maniacal outbursts. As in the first case cited the complaint of pain in the head and paralyzed limbs was a marked feature.

In both of these cases there was evidently a thrombosis, probably in the basal ganglia, if the central pain complained of in the affected limbs can be said to have a localizing value. Recently I[1] studied the subject of central pain and concluded that the most frequent site of the lesion causing this phenomenon was the basal ganglia especially the optic thalamus.

These two cases illustrate a form of psychosis after apoplexy which is I believe of not very frequent occurrence.

It is unlike the dementia post-apoplectic of Ziehen,[2] Bianchi,[3] or the post-apoplectic Schwachsinn of Kraepelin[4]; or the dementia occurring after apoplectic seizures described by Oppenheim,[5] Griessinger[6] and others. These are the usual forms of post-apoplectic mental disturbances and consists of an alteration of the will and the affective faculties associated with a diminution of the intelligence. The patient is restless, irritable, sensitive and shows some change in the character; his memory fails; he is childish; he exhibits shamelessness and other changes in the ethical viewpoint.

My cases resemble somewhat, but still differ from those described by Dupré[7] which exhibit symptoms characterized by excitation, depression, absurd acts, incoherence, demential manifestations, cerebral automatism, incoordination, insomnia, complaints of injury, profanity, incontinence, constituting a *demencé agitie*.

While Dupré and others describe post-apoplectic mental symptoms characterized by delirium, hallucinations, hypochondriacal and depressive mental states with the same characteristics which my cases exhibited, it is not clear that these symptoms constitute an acute curable psychoses; but rather that they are added to or are a part of a progressive and incurable dementia.

It must be understood that both of my cases recovered their normal mental state though perhaps with some simple enfeeblement and certainly not to a full mental vigor, and were therefore not cases of dementia upon which was grafted a delirium, a mania, a hallucinosis or what not but were examples of an acute psychosis of a curable type.

Legrand du Saulle[8] in 1881 gave a very good description of the mental state following apoplectic seizures. He divided the symptoms into three classes.

(1) Those in which the intellectual decline is consistent with a civil and moral responsibility and consists of some change of the character and a weakening of the will; (2) those cases in which the patient is sensitive and emotional with feeble memory and lack of decision and spontaneity, a condition which he says cannot be called either dementia or yet a normal mental state; and (3) those cases in which the patient is more or less disoriented, forgets the identity of the people surrounding him, in which judgment is lost, in fact in which there is a true dementia. This includes also cases in which there are delirium, apprehensions and fears, ideas of persecution, depressive ideas, hallucinations at night and maniacal states, a condition which may go on to a complete dementia.

As far as the symptomatology is concerned my cases fall into the last class described except that no true dementia was at any time manifested by my patients.

Ziehen described a mental state as the result of a thrombosis bearing marked resemblance to the symptoms presented by my cases; namely, disorientation, delirium, perseveration, and particularly a weakening of the attention. He also stated that symptoms of Korsakoff's disease may be manifested.

It will be noted that the delusions in my cases were mildly persecutory in character. This has been noted by Dupré, Lwoff,[9] Legrand du Saulle, Clouston[10] and Magnan,[11] in their descriptions of the delusions of psychosis after apoplexies. Benon[12] observed this type of delusions in 24.1 per cent of his cases.

In my cases there was a hypochondriacal as well as a depressive tinge to the delusions and this has been observed by Ferriere,[13] Dupré, Magnan and Benon.

There was a distinct tendency in these cases to maniacal outbreaks which occurred mainly at night, and were paroxysmal, a condition which had been noted and described by Lwoff, Dupré and Griessinger.

Hallucinations occurred in my cases and persisted throughout the entire course of the disease and were persecutory, phantasmagoric, and depressive in type. The hallucinations occurred during the late afternoon or at night which is characteristic.

They are described by du Saulle as unhappy, persecutory and frightful. In Dupré's experience they consisted of phantoms, animals, terrifying forms and painful hallucinations of smell. The terrifying nature of the hallucinations of sight have been noted also by Charon,[14] and Magnan. Sometimes hallucinations of taste, odor and general sensibility have been observed. (Dupré.) They were presented according to Benon in 17.35 per cent of one hundred cases of hemiplegia.

Position of lesion. Benon has shown that in 51.5 per cent of the cases of post-apoplectic psychoses the palsy in left-sided, and in 48.9 per cent right-sided, therefore practically showing as he believed no preference for either side.

According to Luys,[15] lesions in the left side were almost always accompanied by emotional excitation coexistent with pain in the palsied limbs. This is the only reference to pain in the affected limbs that I was able to find in the literature. It will be remembered that both my patients complained of great pain, spontaneous in character, before any symptoms of psychosis developed and persisted until the mental condition cleared.

Lesions of the first temporal region are said especially to give rise to emotional disturbances (Brissaud,[16] Luys, and Seguin[17]) and softening of the corpora striæ, the insula and the external capsule, Luys believed were also characterized by emotionalism.

Psychic symptoms according to Dupré predominated in alterations of the anterior part of the brain especially in the frontal lobes and in affections of the corpus callosum.

Intellectual disturbances with speech defects was recognized by Hoch as due to implication of the large ganglia and according to Clouston the most frequent site of the lesions in these cases was the basal ganglia. Lesions of the thalamus causes great emotivity according to Bianchi a fact born out by the observations of Holmes and Head[18] and others.

Lesions of the left side of the brain cause defects of intelligence, Bianchi stated, much more than on the right side due to the location of the center of the functions of speech on the left side. Journiac[24] described a case of melancholia with suicidal and homicidal impulses as a result of hemorrhage in the lenticular nucleus without palsy.

Dupré believed that the mental disturbance was proportional to the extent of the lesion and the extent of the cortex implicated.

Ziehen stated that after a thrombosis the intellectual decline is more intense than after embolism or hemorrhage. On the contrary Kraepelin agreed with Mingazini that it made no great difference whether the lesion was a hemorrhage or a thrombosis.

The mental symptoms developing after an apoplexy are looked upon as due to the diffuse vascular lesion of the brain which is associated with the hemorrhage or thrombosis (Ziehen, Charpentier,[19] Ricksher,[20] Paton[21]). Oppenheim called attention to the fact that the dementia was seldom the result of the seizure or caused by the lesion itself merely, but was rather the outcome of this, plus general disease of the cerebral vessels and their consequence, and Bianchi also believed that the arterial sclerosis explained the mental symptoms after cerebral softening.

Autointoxication according to some authorities is the cause of the mental phenomena. Charpentier called attention to this, and Dupré believed that mental confusion with disorientation and anxious states may be associated with hepatic and renal insufficiency which appear to be related to the toxic and infectious nature of the psychic phenomena.

Symptoms of Korsakoff's disease which occurs in some cases, an occurrence described by Ziehen, suggests a toxic origin for this condition.

In these cases it is to be noted that there is in some instances a predisposition (Dupré and Magnan[11]), and the rôle played by heredity (Clouston) and alcohol must not be overlooked (Lwoff).

The prognosis is unfavorable in the usual forms of psychoses described. The dementia is looked upon as being progressive (Ball and Chambard[22] and Benon), and is said to be worse in those cases in which there are foci of softening. Benon believed that the mental troubles rarely ameliorate. On the contrary the cases herein described show that there may occur a form of post-apoplectic psychosis which is recoverable.

A mental state preceding the attack has been described by Savage[22] expressing itself as irritability, emotionalism, poor memory, simple pain in the eyes, and hallucinations of hearing. Ball and Chambard stated that it was rare for the attack not to be preceded by sensory and intellectual troubles for weeks or months due he believed to atheromatous states of the cerebral arteries. A prodromal stage was described by Benon consisting of anterograde amnesia, diminution of attention, modification of character, delirious ideas, excitation, depression or confusion. No mental symptoms preceding the attack were noted in my cases.

Evidence of autointoxication either from renal or hepatic insufficiency was lacking in my cases. There was in the first case described a distinct element of hereditary predisposition and both cases were of an age when arterial sclerosis of the brain could be expected to be present.

In conclusion I wish to emphasize the following features of these cases, namely, the acuteness of the attack; the curability of the psychosis; the presence of pain in the head and in the affected members; the depressive, hypochondriacal trend of the mental symptoms; the presence of perseveration; the occurrence of hallucinations of an unpleasant type and finally maniacal outbursts, the hallucinations and maniacal manifestations being worse at night.

REFERENCES.

1. Rhein: Jour. of Ner. and Men. Dis., 1912, p. 660.
2. Ziehen: Psychiatrie, 1911, p. 310.
3. Bianchi: Text-book of Psychiatry, 1906, p. 846.
4. Kraepelin: Psychiatrie, 1904, Vol. 2, p. 483.
5. Oppenheim: Text-book of Ner. Dis., 1911, Vol. 2.
6. Griessinger: Mental Pathology and Therapeutics, 1882, p. 123.
7. Dupré: Ballet. Traite de Path. Ment., 1903, p. 1065.
8. Legrand du Saulle: Gaz. des Hos., 1881, p. 537.
9. Lwoff: Etude sur les troubles intellectual liés aux lésions Circonscrites du cerveau. These de Paris, 1890.
10. Clouston: Men. Dis., 1904.
11. Magnan: In Dupré.
12. Benon: These de Paris, 1905.
13. Ferriere: These de Paris, 1889.
14. Charon: Arch. de neurol., 1899, Vol. 8, p. 433.
15. Luys: In Ball and Chambard.
16. Brissaud: In Charon.

17. Seguin: In Paton.
18. Holmes and Head: Brain, 1911, p. 255.
19. Charpentier: These de Paris, 1904.
20. Ricksher: Am. Jour. Insanity, 1906, p. 55.
21. Paton: Psychiatry, 1905, p. 546.
22. Ball and Chambard: Dict. des Sciences Médicales, 1st Série, 1882, p. 581.
23. Savage: Jour. of Ment. Science, 1883, p. 90.
24. Journiac: Ann. Médico-Psycholog., 1891, xiii, p. 431.

University of Pennsylvania.
Contributions from the Dept. of Neurology and the Laboratory of Neuropathology.

P
Med
P

146813

Biological
& Medical
Serials

Pennsylvania, University of
Contributions from the Department of
Neurology and the Laboratory of Neuropathology.
Vol.7(1913-14)

University of Toronto
Library

Biological
& Medical
Serials

DO NOT
REMOVE
THE
CARD
FROM
THIS
POCKET

Acme Library Card Pocket
LOWE-MARTIN CO. LIMITED